Pediatric Fundamental Critical Care Support

Third Edition

Society of Critical Care Medicine
The Intensive Care Professionals

Pediatric Fundamental Critical Care Support

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ASSESSMENT OF THE CRITICALLY ILL CHILD

Objectives

- Review anatomic and physiologic differences between pediatric and adult patients.
- Apply the DIRECT methodology — detection, intervention, reassessment, effective communication, and teamwork.
- Recognize respiratory failure and describe the clinical features of the different types of shock.
- Discuss the role of ancillary tests in the cardiopulmonary evaluation of a child.
- Explain how to perform a rapid evaluation of a child's physiological status.
- Apply the Pediatric Early Warning System (PEWS) score to detect clinical deterioration early.
- Discuss the early recognition and treatment of sepsis.

Case Study

An explosion in the local shopping center resulted in multiple injuries. An ambulance arrives at the hospital with 2 patients. The first is a 14-year-old girl who steps out of the vehicle screaming hysterically for someone to help her younger brother on the stretcher. She smells of smoke, her clothes are ripped, and she has a large, profusely bleeding laceration on her forehead. Her 8-year-old brother is unconscious and blood is coming out of both ears. The paramedic is applying direct pressure to try and stem the bleeding from a traumatic amputation of his right arm.

Detection

– Who do you treat first?

– What are the triage priorities in a mass casualty situation?

– What is the boy's mental status?

Intervention

– What equipment do you need? How do you determine the appropriate size for these items?

– What fluids should be administered, and how?

– Would applying a tourniquet be appropriate management of the boy's ongoing blood loss? Could a blood pressure cuff work as a tourniquet?

– Is he a candidate for damage control resuscitation?

– How do you care for the posttraumatic medical and emotional needs of those affected by trauma?

Reassessment

– What is the boy's response to the fluid administration? Is there any response?

– What is his mental status now?

Effective Communication

– What problems and outcomes of particular interventions have to be communicated among team members?

– Which monitoring systems have to be attached and interpreted?

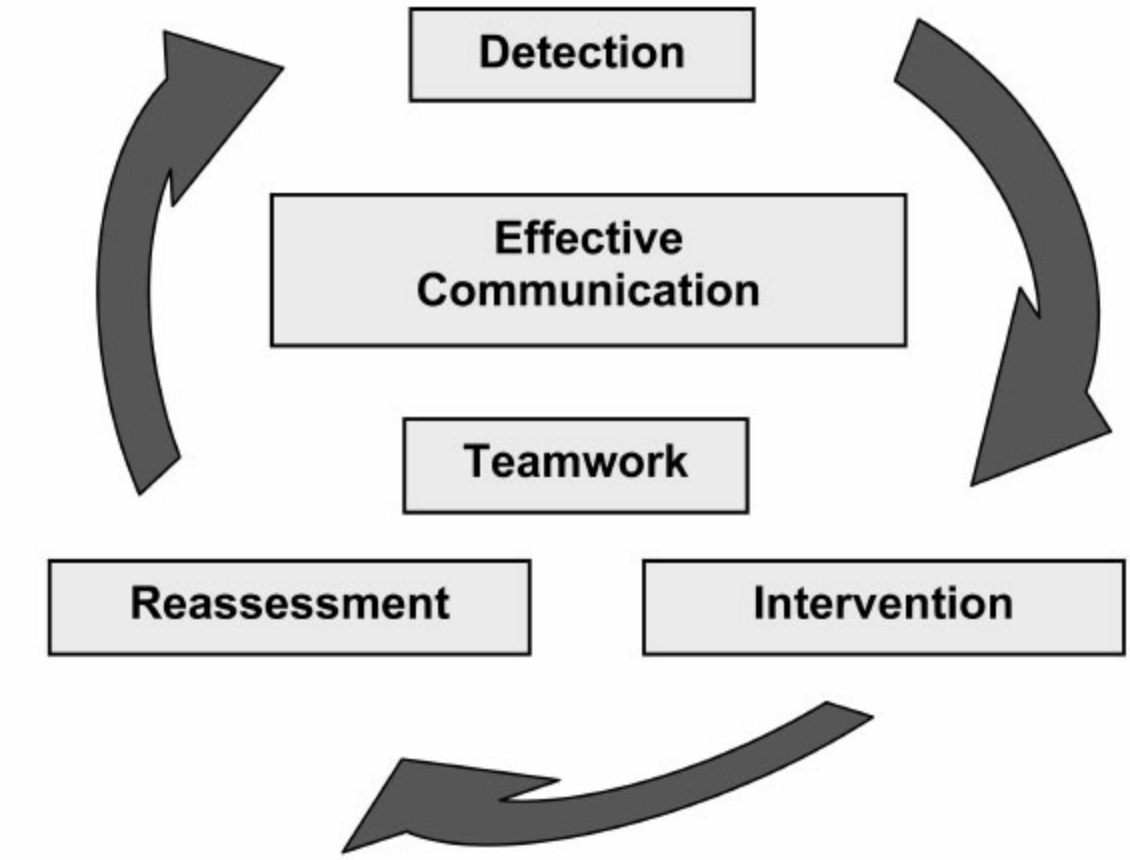
Teamwork

– What medical/surgical specialties must be involved in the care of this patient?

I. INTRODUCTION

This chapter will outline the maturational, anatomic, and physiologic differences of children and their responses to shock, trauma, and illness as contrasted to those of adults. This chapter also introduces the key PFCCS course learning and management concept, **DIRECT**: detection, intervention, reassessment, effective communication, and teamwork (**Figure 1-1**)

Figure 1-1. DIRECT Methodology



Detection: Using the history, physical exam, and Pediatric Early Warning System (PEWS) score alerts the critical care team to the physiological status of the child. These results then guide appropriate laboratory and radiographic evaluations to establish a working/presumptive diagnosis, differential diagnoses, and worst possible diagnoses.

Intervention: This is the process of treating and correcting the disease or injury while keeping in mind the critical care maxim to minimize morbidity and prevent mortality.

Reassessment: This ensures the treatment is appropriate for the severity of the disease and/or injury.

Effective Communication: The greatest source of injury and death in healthcare is communication errors. The more complicated the patient, the more important it is for everyone to communicate their perspectives to the team, so that multiple and often time-sensitive tasks can be done expertly and promptly.

Teamwork: The patient does best when all individuals on the healthcare team bring their specialized training to work together synergistically to care for the needs of the critically ill or injured pediatric patient.

Many steps must be performed in parallel as part of the primary survey (**Table 1-1**). Ideally, the traumatically injured child is evaluated and treated at the closest appropriate facility, but such a decision begins at the scene of injury and is reevaluated continuously along the levels of care. Laboratory investigations and radiologic imaging studies must not delay the evaluation, treatment, or transport of the critically injured child to the center for appropriate definitive care (**Chapter 9**). Damage control resuscitation — a trauma treatment strategy consisting of early hemorrhage control, permissive hypotension, and hemostatic resuscitation — is focused on preventing and reversing the

lethal triad of hypothermia, acidosis, and coagulopathy in the trauma patient.

Table 1-1	PRIMARY SURVEY, ADJUNCTS, AND SECONDARY SURVEY MNEMONICS

Primary Survey: SCABDE
S – Safety: standard precautions, scene safety, patient and parent safety
C – Circulation: stop obvious bleeding (tourniquet) and start volume resuscitation
A – Airway: secure the airway with cervical spine stabilization
B – Breathing: oxygenate and ventilate; apply pulse oximetry and capnography
D – Disability: pupillary response, Glasgow Coma Scale, and point-of-care glucose test
E – Exposure: remove clothes, log roll, look behind cervical collar, keep warm (blankets, warm fluids, radiant warmer, warming blanket)
Adjuncts to the Primary Survey: FFG
F – Foley: after rectal exam to check for high-riding prostate in males, suggestive of urethral injury
F – Focused Assessment Sonography in Trauma (FAST) exam
G – Gastric tube: place orally if unconscious and until face and basilar skull fractures are ruled out
Secondary Survey: HEELPP MEE!
H – History – AMPLE: allergies, medications, past medical history, last meal, and event details
E – Electrocardiographic monitoring: ensure chest leads are placed as soon as possible
E – End-tidal CO_2 monitoring
L – Laboratory tests: minimum prothrombin time/partial prothrombin time, blood typing and cross matching, arterial blood gas
P – Pulse oximetry
P – Photography: plain film radiographs minimum (anterior-posterior chest and pelvis) and computed tomography of head and neck as indicated
M – Medications: tetanus vaccine and/or immunoglobulin depending on vaccination status; antibiotics and other medications as needed
E – Evaluate: examine entire patient
E – Evacuate: consider a transfer if the patient's needs exceed your institution's capabilities

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II. THE GENERAL EXAMINATION

The general examination is probably the most important part of the physical exam. It begins the moment the clinician sees the pediatric patient and is summed up nicely in the answer to this simple but essential question: "Does this child look sick?" Application of PEWS (**Table 1-2**) expedites the evaluation process by focusing on the behavioral, cardiovascular and respiratory status of the pediatric patient. Because young children are unable to verbalize their complaints, evaluation by the

healthcare provider depends upon general and specific features of the examination in addition to information obtained from a parent or guardian. Although many of the early signs of distress are subtle, their recognition can increase the likelihood that timely interventions will be successful and more serious disease progression will be prevented. If healthcare providers initially miss the more elusive signs of illness, they may later assume a child's condition has suddenly deteriorated when, in fact, a seemingly abrupt change reflects an advanced point along a continuum of physiologic compromise. A PEWS score of ≥3 should prompt an escalation in care, such as informing senior staff, increasing the frequency of vital sign checks and clinical assessments, and/or transfer to an ICU. The reference range by pediatric age for heart and respiratory rates are listed in **Table 1-3**. Important considerations in the general examination of a pediatric patient are listed in **Table 1-4**.

Table 1-2 PEDIATRIC EARLY WARNING SYSTEM (PEWS)

	0	1	2	3	Score
Cardiovascular	Pink or capillary refill 1-2 seconds.	Pale or capillary refill 3 seconds.	Gray or capillary refill 4 seconds. Tachycardia of 20 above normal rate.	Gray and mottled or capillary refill ≥5 seconds Tachycardia of 30 above normal rate or bradycardia	
Respiratory	Within established baseline. No retractions Room Air	≥10 above established baseline. Mild Retractions Up to 2L/min or 30%	≥20 above established baseline. Moderate Retractions Up to 4L/min or 40%	≤30 above established baseline Severe Retractions Grunting Up to 5 L/min or 50%	
Behavior	Playing/ Appropriate or Sleeping			Lethargic or Confused Reduced response to Voice or Pain	
Score an additional 2pts for nebulizer use, suctioning, or persistent vomiting after surgery.					
				Total	

Reproduced from [CC BY-SA 4.0 (https://creativecommons.org/licenses/by-sa/4.0)] from Wikimedia Commons. *Score by starting with the most severe parameters. Score 2 extra for every 15-minute nebulization (including continuous) or persistent postoperative vomiting. Use L/min to score regular nasal cannula. Use FIO_2 to score high-flow nasal cannula.

Table 1-3 PEDIATRIC REFERENCE RANGES FOR HEART AND RESPIRATORY RATES

	Age	**Respiratory rate at rest (breaths/min)**	**Heart rate at rest (beats/min)**
Newborn	Birth - 1 mo	40-60	100-180
Infant	1 - 12 mo	35-40	100-180
Toddler	13 mo - 3 y	25-30	70-110
Preschool	4 - 6 y	21-23	70-110
School age	7 - 12 y	19-21	70-110
Adolescent	13 - 19 y	16-18	55-90

Table 1-4	GENERAL EXAMINATION: ABNORMAL SIGNS

Skin

- Loss of normal pink coloration of mucosa and nail beds
- Mottling
- Skin warmth or coolness
- Prolonged capillary refill, determined with extremity above the level of the patient's heart to prevent mistaken assessment of venous refill

Breathing

- Bradypnea/tachypnea
- Stridor
- Audible wheezing
- Nasal flaring/grunting
- Intercostal retractions

Signs of Dehydration

- Sunken fontanelle in infants
- Absent tears
- Sunken eyes
- Skin tenting
- Dry mucous membranes

Level of Alertness

- Awake and alert
- Responds to verbal commands
- Responds only to painful stimulation
- Unresponsive

Color

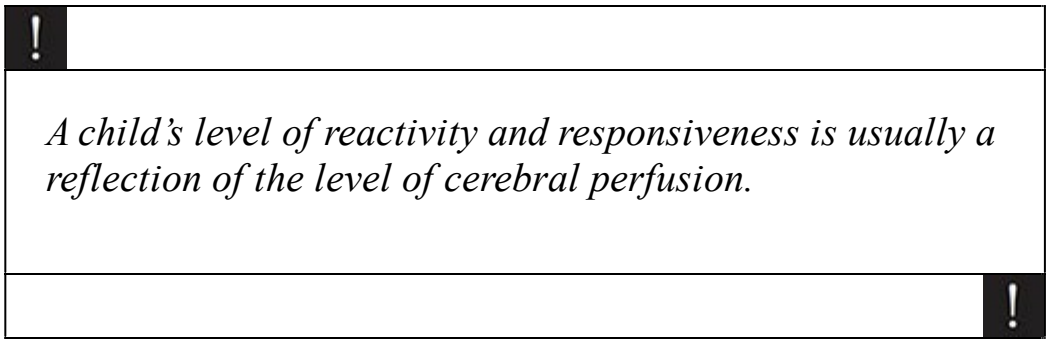
- Cyanosis
- Acrocyanosis (may be present if room temperature is cool)
- Central cyanosis
- Jaundice
- Pallor

During the clinical exam, children should be allowed to remain in the position they spontaneously assume for comfort. For children younger than 1 year, this is often in the arms of a parent or primary caregiver. Illness may be marked by a child's inability to find a position of comfort. Pediatric patients should not be forced to assume a different position than the one they decide upon; doing so could potentially compromise a tenuous airway, as in epiglottitis, severe laryngotracheobronchitis (croup), or foreign body obstruction.

A child's general appearance will immediately help the observer discern the presence of serious illness.

An ill child may initially have increased irritability, followed by decreasing responsiveness, increasing flaccidity, and lethargy. This is observed in conditions such as hypoxia, hypercarbia, uncompensated shock, traumatic brain injury, and hypoglycemia. Depressed mental status requires rapid evaluation of the respiratory and cardiovascular status. In children, respiratory failure most

often precedes cardiovascular failure in the absence of an underlying congenital heart defect or trauma. Signs of physical injury can be very subtle to nonexistent, especially in cases of abusive trauma. In most infants, alertness can be evaluated by observing their ability to fixate on objects, particularly a parent's face. The infant should turn toward sound and should follow an object horizontally and, starting at 1 month of age, should follow an object vertically as well. Older children, 8 months to 2 years, should exhibit anxiety toward strangers and show clear recognition of their parents or caregivers.

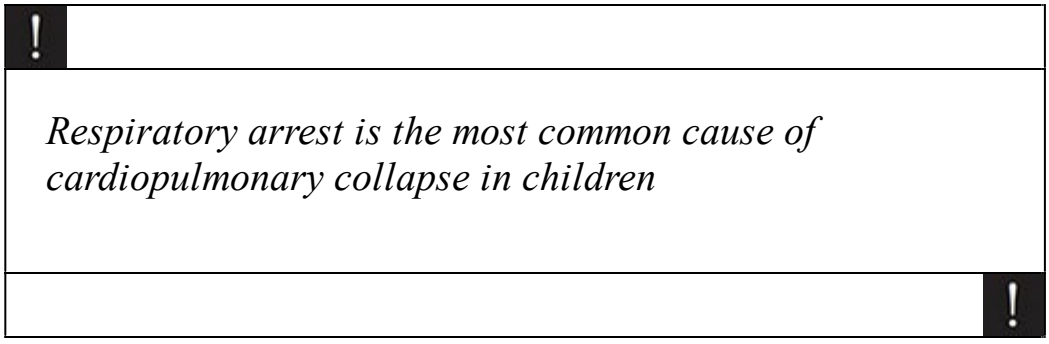


III. RESPIRATORY SYSTEM AND AIRWAY

Respiratory failure is particularly common in infants due to variable maturation in 3 areas:

- Central nervous system receptor and effector mechanisms for the respiratory drive
- Chest wall stability and respiratory muscular strength
- Conducting airways and the alveolar-capillary complex

The respiratory response to hypoxemia in neonates may be biphasic — hyperpnea initially, followed by hypopnea and hypoventilation. This response occurs despite apparently normal central and peripheral chemoreceptors to oxygen and carbon dioxide. It is potentially preventable by early detection and intervention at the initiation of airway and breathing compromise.



A. Anatomic and Physiologic Considerations

The thorax is more cartilaginous in infants and young children and, therefore, is more compliant than in an adult. Inspiration is normally effortless. But when airway resistance is high, the child needs to

generate greater negative intrathoracic pressure to inflate the lungs. The compliant chest wall with soft cartilaginous bones and weak intercostal muscles, retracts inwards under the increased negative intrathoracic pressure. This results in reduced tidal volume and increases the work of breathing and soft tissue retractions. The horizontal muscular insertion points of the pediatric diaphragm on the thorax resemble those of an adult with obstructive lung disease and its associated flattened diaphragm. Therefore, the lower thorax may be drawn inward during inspiration, causing reduced inspiratory volume. Immature intercostal muscles cannot assist active ventilation for several years after birth; thus, more dependency is placed upon diaphragmatic function and excursion. Compromise of diaphragmatic excursion by gastric distention, abdominal distension, surgery and other factors may quickly evolve into impaired respiratory function.

Pediatric lungs are not fully mature until 6 to 8 years of age. Alveolar size and number increase substantially during childhood; lung compliance also increases. Tidal volume as a fraction of total lung capacity remains fairly constant through childhood at 6 to 8 mL/kg of ideal body weight. High intrinsic or externally supplied flow rates are required to deliver this tidal volume because children have a short inspiratory duration. Smaller anatomic conducting airways may produce high resistance if further narrowed by inflammation, edema, mucus, bronchospasm, bronchiolitis, and other conditions. Poiseuille's law states that resistance in an airway is inversely related to its radius to the fourth power, so reducing the airway radius by 50% would increase the resistance sixteen times — $1/(1/2)^4$ (**Chapter 2, Figure 2-1**). Such high peripheral airway resistance may also alter exhalation and induce dynamic closure of the airways and auto–positive end-expiratory pressure. Closing capacity in infants and small children is within their functional residual capacity, thereby making them more susceptible to airway closure during normal breathing. These factors combine to produce less respiratory reserve in the pediatric patient. Consequently, children may decompensate rapidly, leading to cardiopulmonary arrest if there is no intervention.

The majority of deaths in children (especially those younger than 1 year) involve respiratory disorders resulting from infection, poisonings, trauma, submersion, suffocation, or sudden infant death syndrome.

B. Physical Examination

Nasal flaring (increases the nares radii to reduce resistance to the fourth power), tachypnea, grunting (exhalation against a partially closed glottis to prevent collapse of alveoli), and retractions are signs of increased work of breathing as the patient attempts to maintain adequate minute ventilation and oxygenation in response to pulmonary or airway compromise. Given the compliant nature of a child's chest wall, retractions can be profound, with evidence of subcostal, intercostal, and suprasternal retractions. Tachypnea is an important sign of illness in infants and young children, and bradypnea is an ominous precursor of impending respiratory arrest.

Bradypnea is an ominous precursor of impending respiratory failure.

Such extreme variations in respiratory rate have many etiologies (**Table 1-5**). The normal respiratory rate of an infant or child is age dependent (**Appendix 1**). Always be wary of "normal" respiratory rates in a lethargic or poorly responsive pediatric patient. The pediatric brain is more metabolically active than the adult brain. Mental status is a sensitive indicator of inadequate oxygenation, ventilation, and perfusion. As previously mentioned, a patient's position of comfort is usually one allowing adequate gas exchange. For example, a child with upper airway obstruction (eg, epiglottitis) will usually attempt to sit forward or assume a tripod position. According to Poiseuille's law, lower airflow velocity and less viscous inspired air promote laminar airflow, not turbulent airflow, thereby decreasing airflow resistance and subsequent work of breathing. If a child becomes agitated when separated from the parent to facilitate clinical examination, the increase in velocity of airflow (ie, crying) can lead to turbulent airflow, thereby increasing airway resistance and possibly converting a partially obstructed airway (eg, epiglottitis or foreign body obstruction) into a completely obstructed airway.

Table 1-5 CAUSES OF VARIATIONS IN RESPIRATORY RATE

Tachypnea	Bradypnea
• Fever	• Hypothermia
• Pain and anxiety	• Central nervous system injury
• Hypovolemia	• Drug-induced depression
• Respiratory disease	• Neuromuscular disease
• Metabolic acidosis	• Severe shock
• Heart failure	• Metabolic disorders
• Adverse drug effect	
• Hyperviscosity syndromes	

The shape and movement of the chest during respiration will alert the observer to underlying respiratory problems. A rib-cage deformity, such as pectus excavatum, pectus carinatum, or scoliosis, may suggest the presence of restrictive lung disease, pulmonary hypoplasia, and/or abnormal respiratory mechanics. Asymmetry of chest rise can be noted visually and by laying one's palms on each side of the patient's chest and noting differential hand rise. Asymmetry is indicative of unequal air entry and serious underlying pathology (**Table 1-6**). Auscultation of breath sounds will reveal the adequacy and symmetry of air entry and allow detection of any other sounds, such as wheezes, crackles, and rubs. Auscultation may be difficult or easy, depending on the child's level of cooperation and environmental surroundings. Because children have thinner chest walls than adults, breath sounds are easily audible but less easily localized, and other transmitted sounds often interfere with accurate auscultation. When checking for symmetric breath sounds after intubation, it is best to auscultate in the anterior axillary line as opposed to the midclavicular line to minimize contralateral breath sound transmission and false identification of equal breath sounds bilaterally. Nevertheless, when a child is agitated and crying, adequate auscultation may not be possible.

Respiratory embarrassment is manifested by nasal flaring,

tachypnea, grunting, and retractions.

!

Table 1-6	CAUSES OF ASYMMETRICAL CHEST MOVEMENT
	Tachypnea
	• Unilateral pneumothorax • Unilateral pleural effusion • Foreign body aspiration with hyperinflation • Mucous plugging of main-stem bronchus • Lobar atelectasis

In general, cyanosis of the skin and nail beds, if present, is indicative of hypoxemia, but may be a late finding in the hypoxemic child. Cyanosis is dependent upon the patient's total hemoglobin concentration because >5 g/dL must be desaturated for cyanosis to be clinically observed. Because children have lower hemoglobin concentration than adults or in the setting of acute blood loss, the blood oxygen content must fall to very low levels before cyanosis is clinically evident. Oxygenation is dependent upon the mean airway pressure and the percentage of inspired oxygen.

!

Cyanosis is a poor indicator of hypoxemia in infants and children.

!

The advent of pulse oximetry has enabled the noninvasive measurement of hemoglobin oxygen saturation. Pulse oximetry does not accurately assess ventilation status, but end-tidal CO_2 capnography does. There are several advantages of capnography:

- Allows noninvasive monitoring of ventilation
- Provides measure of respiratory rate and regularity of breathing
- Confirms and continuously monitors endotracheal tube placement in trachea
- Sounds alarms first when endotracheal tube becomes dislodged or obstructed
- Correlates alveolar CO_2 with arterial CO_2
- Detects lower airway obstruction with waveform

!

Children in respiratory distress should be allowed to choose their position of comfort.

Alveolar ventilation is primarily responsible for removal of carbon dioxide and is represented by minute ventilation, which is the product of the respiratory rate and tidal volume.

A patient may maintain adequate oxygen saturation in the face of inadequate minute ventilation, especially if supplemental oxygen is provided. Therefore, clinical assessment, including attention to mental status, chest movement, respiratory rate, work of breathing, and capnography, is crucial to determine the adequacy of CO_2 minute ventilation. Another essential respiratory monitoring adjunct to the clinical examination is arterial blood gas sampling, as well as transcutaneous CO_2 monitoring if available.

Minute ventilation = respiratory rate x tidal volume

C. Airway Evaluation

Case Study

A 20-month-old infant, born prematurely at 24 weeks, presents with yet another episode of "croup" (ie, laryngotracheobronchitis) despite having received respiratory syncytial virus immunoglobulin. He has a history of multiple and prolonged intubations. He has always been a noisy breather, but now is irritable, won't eat much, and is receiving 35% oxygen at a rate of 2 L/min. He looks pale and has a capillary refill of 3 seconds and respiratory rate of 45 breaths/min. A chest radiograph shows his lungs are clear and his heart is normal size. His parents are wondering why this keeps happening.

A systematic approach to the patient's signs and symptoms would help DIRECT this patient's care.

Detection

– Why is this patient having recurrent laryngotracheobronchitis?

– What is his PEWS score?

– What noninvasive monitoring might be useful in evaluating this patient?

– What additional films might be helpful?

Intervention

– Will bronchodilators be of benefit?

– What might help keep the child calm?

Reassessment

– Is bronchoscopy/laryngoscopy the best initial course of action?

Effective Communication

– It is important for all team members to keep the child calm and assume the position of comfort.

Teamwork

– How can the team stabilize the child's condition until the airway can be evaluated safely?

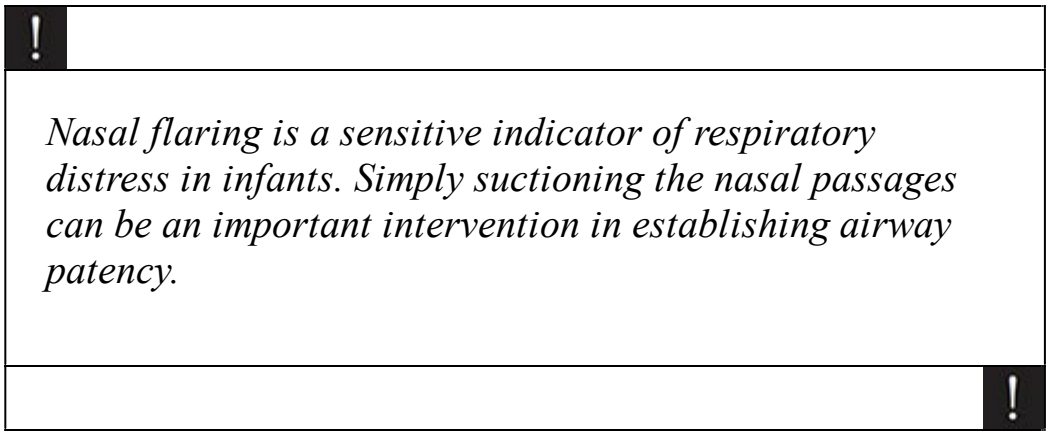
In this case, the patient's PEWS score is: behavior irritable = 2, cardiovascular capillary refill 3 seconds = 1, respiratory rate >10 above normal = 1, for a total = 4. This is a case of subglottic stenosis caused by prolonged intubation of a premature infant, resulting in airway obstruction and respiratory compromise. These same signs and symptoms have been misinterpreted as indicators of laryngotracheobronchitis and can present as a life-threatening airway emergency.

The first consideration is to allow the pediatric patient to find a position of comfort. A child with impending airway obstruction (eg, foreign body ingestion or epiglottitis) often prefers to sit forward leaning on the outstretched arms (ie, tripod position). If the child is unconscious or obtunded, head position is paramount. In the setting of trauma, stabilization of the cervical spine with immobilization – to avoid incidental movement, hyperextension, or flexion – is essential to decrease the risk of iatrogenic spinal cord injury. The child younger than 2 years has a relatively large occiput which causes the neck to flex forward, potentially obstructing the airway and/or further exacerbating a cervical spine injury, when the child is placed on a firm mattress or rigid backboard. The sniffing position is preferred and can be accomplished by tilting the head back slightly and/or placing a rolled towel beneath the shoulders in those younger than 2 years. A folded towel or sheet under the child's occiput can accomplish the sniffing position in patients older than 2 years. Infants, particularly those younger than 2 months are obligate nasal breathers. Simple suctioning is often an important intervention to relieve an airway obstruction, especially in an infant with respiratory syncytial virus infection. In the case of congenital choanal atresia, the neonate has difficulty eating and is cyanotic at rest but pink when crying. An oral airway and/or prone positioning may be all that is needed to maintain an open airway at rest until the anomaly can be surgically corrected. In the setting of trauma, a length-based weight, equipment, and emergency medications tape (eg, Broselow tape) are invaluable in quickly ascertaining the appropriately sized equipment and medication doses needed to assist intubation. Exercise caution regarding the quality and clinical accuracy of smartphone applications for pediatric medication dosing and treatment protocols.

The first intervention for respiratory compromise (cyanosis, tachypnea, nasal flaring, and chest wall retractions) is to administer oxygen. This can be done using any number of devices: non-rebreathing mask, face mask, air-entrainment (Venturi mask), nasal cannula, oxygen hood, or face tent (**Chapter**

2). Pulse oximetry and arterial blood gas measurements can be used to assess for an adequate or excessive amount of oxygen supplementation. Capillary blood gas measurements approximate arterial blood gas pH and arterial CO_2 levels, but do not provide an accurate arterial oxygen concentration. Hypoxia and hyperoxia are deleterious and should be avoided.

A child has 2 to 3 times the oxygen consumption rate and resting energy expenditure of an adult. When a child presents in respiratory distress, one should first administer the highest possible percentage of oxygen using a non-rebreathing face mask, then titrate down the percentage of oxygen delivered. The usual arterial oxygenation goal is 80 to 100 mm Hg on arterial blood gas analysis, which corresponds approximately to a pulse oximetry of 94% to 98%. Using the most appropriate and best tolerated oxygen delivery device, supplemental oxygen ought to be heated and humidified to avoid drying the mucosa of the oro- and nasopharynx. Airway obstruction, aspiration, and apnea are serious hazards to respiratory function.


!
Nasal flaring is a sensitive indicator of respiratory distress in infants. Simply suctioning the nasal passages can be an important intervention in establishing airway patency.
!

The anatomy of the pediatric airway, especially in infants and young children, predisposes these patients to airway obstruction when they are positioned incorrectly. In addition to a large occiput, children younger than 2 years have a relatively large tongue, a larger and more floppy epiglottis, and an anteriorly placed larynx. These factors, combined with decreased hypopharyngeal tone, lead to the loss of airway patency in obtunded patients because they are unable to keep these soft tissue structures apart. (See **Chapter 2** for the factors affecting pediatric endotracheal intubation.)

In young children, common causes of airway obstruction are viral croup, bacterial tracheitis, foreign body, and, less commonly, epiglottitis in countries with successful *Haemophilus influenza* type B immunization programs. Clinical examination may help identify the site of obstruction. Airway obstruction above the thoracic inlet tends to cause stridor (inspiratory noise), as in viral croup or post-extubation laryngeal edema. These stridulous patients may respond to nebulized racemic epinephrine (adrenaline, 0.05 mg/kg; maximum 0.5 mg in 3 mL normal saline) and IV steroids (dexamethasone, 0.5 mg/kg; maximum dose, 10 mg every 6 h).

Asthma is not the sole cause of wheezing and intrathoracic lower airway obstruction. Infants with respiratory syncytial virus bronchiolitis may present similar symptoms. Children wheezing from asthma should receive a combination of supplemental oxygen, inhaled β-agonist, corticosteroid and ipratropium bromide. Children with both upper and lower airway obstruction and low oxygen requirements (eg, asthma, bronchiolitis) may benefit from improved laminar flow through the narrowed passages by breathing heliox, a mixture of oxygen and helium (typically 30% oxygen and 70% helium). The less dense the inhaled mixture, the less likely it is to have obstructive turbulent

flow at higher velocities through disease-narrowed airway passages.

If there is no response to these therapies, then the presence of foreign body or vascular ring obstruction must be entertained. Vascular rings, (the aorta and/or its branches encircle the trachea) usually present between 2 and 5 years of age. Foreign bodies do not allow the affected lung to deflate on exhalation due to the ball-valve action, whereby air can get in past the obstruction but can't get out. Both foreign body aspiration and vascular rings need a high index of suspicion in the pediatric age group to make the correct diagnosis.

D. Respiratory Failure

Children in respiratory distress progressing to respiratory failure will exhibit increased work of breathing until they experience muscle fatigue or are no longer able to compensate. Causes of respiratory failure in children can usually be grouped by age. In premature neonates, respiratory failure results from apnea of prematurity or neonatal respiratory distress syndrome, in which atelectasis and impaired gas exchange occur as a result of surfactant deficiency and ineffective chest bellows. In term neonates, bacterial pneumonia, sepsis, meconium aspiration, and congenital airway abnormalities are most common. In infants and toddlers, lower respiratory disease from pneumonia, bronchiolitis, asthma, foreign-body aspiration, and upper airway obstruction related to infection are common. The causes of respiratory failure in older children are similar to those found in adults (eg, drug overdose, foreign body obstruction, chemical/smoke inhalation, infections). Treatment of respiratory failure begins with early recognition and securing the airway. All necessary equipment must be available and checked for function to facilitate a safe and successful intubation (**Chapter 2**).

Noninvasive monitoring has become indispensable in the evaluation of patients in respiratory distress. With pulse oximetry and end-tidal CO_2 capnography, oxygenation and ventilation can be objectively monitored. Ancillary investigative tests, such as chest radiograph, arterial blood gas examination, and other studies summarized in **Table 1-7**, are useful in confirming respiratory failure, elucidating its etiology, and monitoring response to therapy. These tools are not substitutes for clinical examination, however. The clinical assessment and judgment of the healthcare provider are the most valuable tools for recognizing a patient in impending respiratory failure.

Table 1-7 ANCILLARY STUDIES TO EVALUATE RESPIRATORY STATUS

Study	Evaluation
Arterial blood gas (room air)	Hypoxemia = low Pao_2 (<60 mm Hg [<8.0 kPa])
	Hypercarbia = high $Paco_2$ (>45 mm Hg [>6.0 kPa])
	Acidosis = pH <7.35
	Alkalosis = pH >7.45
	Adequate Pao_2 does not indicate adequate oxygenation if hemoglobin is low pH >7.35 and $Paco_2$ 35-45 mm Hg in a child with severe increased work of breathing is sign of impending respiratory failure
Pulse oximetry (Spo_2)	Noninvasive measure of oxyhemoglobin
	Goal: 94%-98% when supplemental oxygen is used

	Monitor oxygenation in response to treatment; avoid hypoxia and hyperoxia
Hemoglobin	Cao_2 = (Hgb x 1.34 x Sao_2) + dissolved oxygen (0.0031 x Pao_2)
	Do_2 = Cao_2 x cardiac output
Capnography	Measures exhaled CO_2 from nasal cannula, ETT, or tracheostomy tube
	Noninvasively monitors adequacy of ventilation and alveolar CO_2 as it correlates with arterial CO_2
	Confirms and monitors tracheal placement of ETT
	Detects lower airway obstruction with waveform appearance
	Determines physiological dead space
Peak expiratory flow rate	Generates maximum flow during forced exhalation
	Decreases with lower airway obstruction (asthma)
	Monitors bronchodilator therapy
	Requires cooperation to perform, and therefore may be useful only in older children
Chest radiograph	Airway obstruction
	Pneumothorax and/or pleural effusion
	Parenchymal lung disease
	Atelectasis
	Volutrauma

Hgb, hemoglobin; ETT, endotracheal tube; Cao_2, concentration determinant of arterial oxygen content; Sao_2, arterial oxygen percent saturation; Do_2, oxygen delivery

E. Mechanical Ventilation

When supporting oxygenation and ventilation with positive pressure bag-valve-mask or a mechanical ventilator, volutrauma – damage to a lung caused by lung overdistention – must be avoided. It is very easy to hypo- or hyperventilate a patient. Close attention to chest rise can ensure adequate, not excessive, chest expansion during bag-valve-mask/endotracheal tube ventilator support. When selecting the tidal volume for mechanical ventilation, the ideal body weight should be used, especially if the patient is obese. Furthermore, it is important to avoid volutrauma by first selecting a lower volume and advancing to higher volumes as dictated by chest rise, auscultation of breath sounds from superior to inferior lobes, and clinical improvement. The typical tidal volume for a pediatric patient will range from 6 to 8 mL/kg. Conventional mechanical ventilation is mainly delivered by pressure or volume ventilation. Traditionally, pressure ventilation was used in small children and neonates because the mechanical ventilators at the time could not deliver small tidal volumes accurately. With miniaturization and advances in technology, ventilators are capable of delivering tidal volumes as low as 3 mL for use in extremely premature infants (**Table 1-8**). Furthermore, the line between pressure and volume ventilation has been blurred in new modes of mechanical ventilation (eg, pressure-regulated volume control) to take advantage of their differences in the hopes of minimizing ventilator-induced lung injury (**Chapter 5**).

Table 1-8	INITIAL MECHANICAL VENTILATOR SETTINGS FOR INFANTS WEIGHING <5 kg
Initial mode	**Choose between volume-controlled and pressure-limited settings**
Tidal volume	4-6 mL/kg; set directly based on ideal body weight in volume-controlled setting and indirectly by selecting peak inspiratory pressure limit, usually 20-24 cm H_2O pressure
Time limit	Inspiratory time: neonates, 0.25-0.4 s; infants, 0.5-0.6 s; less compliant lungs can tolerate longer inspiratory time as the exhalation time constant in stiff lungs is short
Respiratory rate	30-40 breaths/min as a back-up rate in case of apnea
Positive end-expiratory pressure	5-7 cm H_2O to prevent atelectasis at end of exhalation

In pediatric patients, the respiratory system receives most of the critical care attention because most cardiovascular failure is preceded by respiratory failure. It is vitally important to detect, intervene, and reassess the pediatric airway continuously and to communicate effectively as a team to minimize pediatric morbidity and to prevent mortality.

IV. CARDIOVASCULAR SYSTEM

A. Anatomic and Physiologic Considerations

The circulating blood volume per kilogram is higher in children than in adults, but the absolute volume is lower in children due to their small body size. Consequently, children are less able to tolerate small amounts of blood loss compared to adults. Blood replacement is dependent upon the patient's clinical status, vital signs, ongoing blood loss, risks of transfusing packed red blood cells, and current hematocrit relative to expected hematocrit (**Table 1-9**).

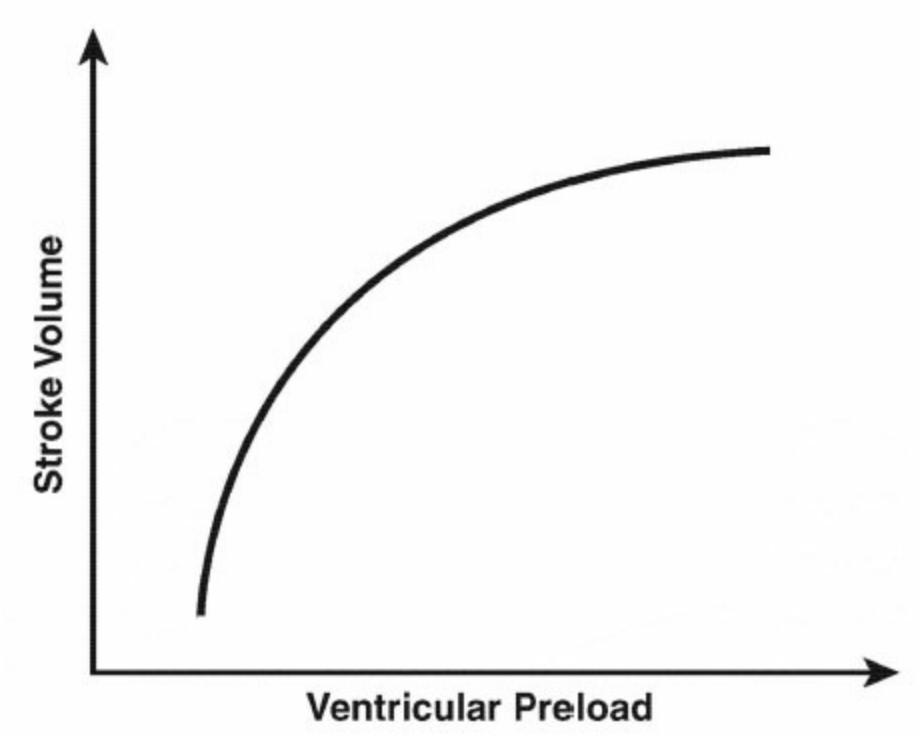
Table 1-9	PEDIATRIC HEMOGLOBIN/HEMATOCRIT VALUES BY AGE AND GENDER	
	Hematocrit Calculated from mean corpuscular volume and red blood cell count (electronic displacement or laser)	
Age	**Reference values (conventional)** % of packed red cells (red cells/whole blood cells × 100)	**Reference values (SI)** Volume fraction (red cells/whole blood)
0-30 d	44%-70%	0.44-0.70
1-23 mo	32%-42%	0.32-0.42
2-9 y	33%-43%	0.33-0.43
10-17 y (male)	36%-47%	0.36-0.47
10-17 y (female)	35%-45%	0.35-0.45
>18-99 y (male)	42%-52%	0.42-0.52
>18-99 y (female)	37%-47%	0.37-0.47

Age	Hemoglobin g/dL	mmol/L
0-30 d	15.0-24.0	2.32-3.72
1-23 mo	10.5-14.0	1.63-2.17
2-9 y	11.5-14.5	1.78-2.25
10-17 y (male)	12.5-16.1	1.93-2.50
10-17 y (female)	12.0-15.0	1.86-2.32
>18-99 y (male)	13.5-18.0	2.09-2.79
>18-99 y (female)	12.5-16.0	1.93-2.48

SI, International System of Units
Information taken from Kliegman RM, Nelson WE, et al. *Nelson Textbook of Pediatrics*. Philadelphia: Elsevier Saunders; 2016.

Cardiac output is dependent upon heart rate and stroke volume, as shown in the formula: cardiac output = heart rate x stroke volume. Stroke volume is dependent upon preload, contractility, and afterload. The ability of the heart to change its force of contraction in response to changes in preload (venous return) is described by the Frank-Starling curve (**Figure 1-2**).

Figure 1-2. Frank-Starling Curve: Relationship of Myocardial Force of Contraction and Filling Pressure



Children, especially infants, have increased vagal tone. When a laryngoscope touches the posterior pharynx or an endotracheal tube suction catheter contacts the pediatric carina, the vagus nerve is stimulated, which innervates the heart's atrial-ventricular conduction node. This increased vagal nerve parasympathetic discharge can precipitate a rapid decline in heart rate, resulting in a dramatic decrease in cardiac output, which may precipitate cardiac arrest. Appropriate use of atropine before intubation and care to avoid passing the endotracheal tube suction catheter beyond the end of the endotracheal tube will minimize this risk.

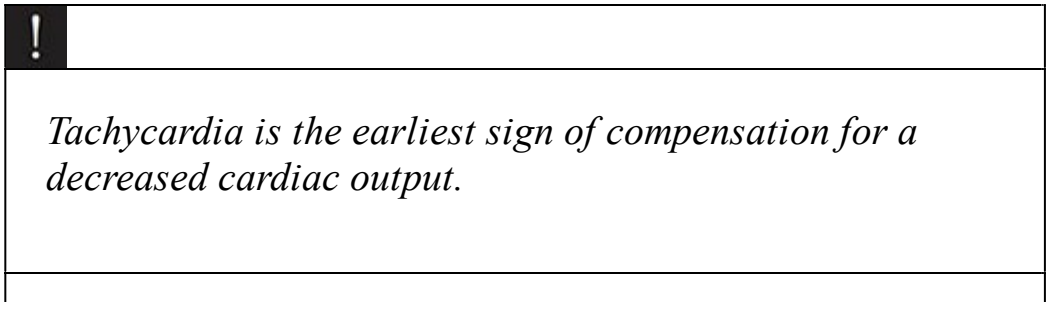
The myocardium of an infant younger than 8 weeks has limited ability to stretch, thereby limiting his/her ability to increase cardiac output in response to increased preload from blood or fluid boluses. The fetal right heart is dominant in utero and is responsible for approximately 60% of the systemic cardiac output through the ductus arteriosus. After birth, the lungs expand and the ductus arteriosus gradually closes, typically over 2 days. Right-sided resistance gradually falls to adult levels by 8 weeks of age. Newborn myocardial anatomy changes so that the initially larger right ventricle decreases in mass and the left ventricle increases in size and mass. Illness, hypoxemia, hypothermia, acidosis, or congenital heart defects may interrupt this physiological transition by maintaining elevated pulmonary blood pressure and a patent ductus arteriosus.

In neonates, exercise caution regarding central venous pressure monitoring interpretation because it does not necessarily reflect circulatory blood volume or left ventricular efficiency. Left and right heart function may be disparate, and each side may fail independently. Therefore, pulmonary artery catheters may occasionally be required to monitor left and right heart filling pressures. The neonatal myocardium exhibits a limited response to catecholamines due to immaturity of sympathetic nervous system innervation, as well as a reduced number of β_1-receptors, both of which increase over the first several weeks of life. Thus, the physiologic effects of exogenous catecholamine administration may be quite variable; careful titration to the individual infant's response is essential.

Any augmentation of pediatric cardiac output is dependent upon the patient's heart rate and diastolic filling time. The absolute amount of cardiac output is small (~600 mL/min); however, the cardiac output per kilogram of weight in the newborn (>200 mL/kg/min) is higher than that of adults, with the cardiac index at birth (4 L/min/m²) gradually falling to adult normative values (2.5 to 3 L/min/m²) in adolescence. This occurs because oxygen consumption is dependent on cardiac output, and oxygen consumption per kilogram of body weight is greater in infants than in adults. Familiarity with the normative ranges of heart rates is important because those ranges vary with age (**Appendix 1**). Rarely do arrhythmias produce significant changes in cardiac output, with the exception of sustained supraventricular tachycardia. Severe tachycardia will decrease diastolic filling time, resulting in a rapid fall in stroke volume. Ventricular arrhythmias are uncommon, but may signify congenital heart disease, myocarditis, cardiomyopathy, or electrolyte imbalance when present. Bradycardia will result in decreased cardiac output and therefore decreased oxygen delivery, and is most often an ominous sign of significant hypoxemia or acidosis.

B. Physical Examination

The physical examination of the cardiovascular system should focus on assessing tissue perfusion as it relates to circulation and end-organ function. This includes evaluation of mental status, skin color, temperature, capillary refill, pulse character, heart rate, rhythm, and blood pressure.



Cardiac output in children, especially infants, is dependent on the patient's heart rate. Blood pressure varies with age, height, and gender. Hypotension can be defined as a systolic blood pressure or mean arterial blood pressure below the fifth percentile. In children aged 1 to 10 years who are at the 50th percentile for height, the predicted fifth percentile systolic blood pressure and mean arterial blood pressure can be quickly determined using the following formulas:

$$\text{Systolic blood pressure (mm Hg)} \quad 70 + (\text{age in years} \times 2)$$

$$\text{Mean arterial pressure (mm Hg)} \quad 40 + (\text{age in years} \times 1.5)$$

The end-organ systems of special relevance are the brain and the kidneys because the body's homeostatic mechanisms give priority to preserving perfusion to these tissues. Brain and kidney function are assessed by evaluating the patient's mental status and urine output. Deterioration of end-organ function is a precursor to circulatory arrest. In addition to the clinical exam, several ancillary studies (**Table 1-10**) should be used to fully evaluate and monitor the critically ill child.

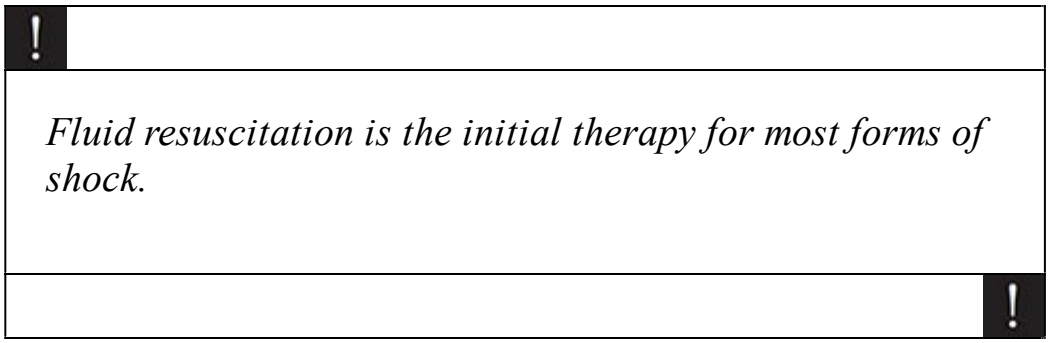
Table 1-10 ANCILLARY STUDIES TO EVALUATE CARDIOVASCULAR SYSTEM

Study	Evaluation
Blood gas	May use both arterial and venous blood gases if necessary Detects acid-base imbalances
Serum lactate	Forms as result of tissue hypoxia and anaerobic metabolism Reflects poor tissue perfusion Serves as a prognostic indicator to assess response to therapy
Total serum CO_2	Measures all forms of CO_2 (primarily bicarbonate) Measures severity of metabolic acidosis
Hemoglobin concentration	Gauges arterial oxygen content and therefore oxygen delivery Monitors ongoing blood loss in hemorrhagic shock
Urinary catheter	Continuously monitors urine output Children: 1-2 mL/kg/h Adolescents: 0.5-1 mL/kg/h
Continuous arterial pressure monitoring	Provides ongoing evaluation of blood pressure Supplies information on systemic vascular resistance and cardiac output through waveform analysis Monitors fluid resuscitation and inotropic/vasopressor therapy Facilitates arterial blood gas sampling
Central venous pressure monitoring	Assesses preload and guides fluid therapy Increases in obstructive forms of shock Measures central venous oxygen saturation Evaluates cardiac output and oxygen delivery
Chest radiograph	Assesses for congestive heart failure, cardiomegaly, pulmonary edema, pleural effusion
Echocardiography	Shows contractility

	Measures estimated ventricular pressures Displays chamber size and wall thickness Demonstrates congenital anomalies Shows pericardial effusion

C. Shock

Shock may be defined as a state of cardiopulmonary dysfunction that fails to provide sufficient oxygen and nutrients to meet the metabolic needs of vital organs and peripheral tissues. For example, when a patient with hemorrhage continues to lose blood, the body compensates by trying to maintain adequate perfusion pressure to essential organs. The heart rate increases in an attempt to maintain adequate cardiac output as venous return diminishes, along with a compensatory increase in systemic vascular resistance. This results in preservation of a normal blood pressure during the early (compensated) phase of shock, with hypotension occurring when the circulatory compensatory mechanisms are overwhelmed by uncorrected, ongoing blood loss. When hypotension is present, the patient is in late-stage (decompensated) shock, which is accompanied by weak, thready pulses, markedly prolonged capillary refill time, mottled extremities, and obtunded sensorium. Timely recognition of the shock state and aggressive intervention are essential to obtaining an optimal outcome. As soon as the diagnosis of shock is considered, early vascular access, treatment and cardiopulmonary monitoring must be implemented.



Rapid restitution of circulating intravascular volume is critical if end-organ damage is to be avoided. Hepatomegaly can be a sign of fluid overload in the pediatric patient; however, it must be viewed with caution. Disease processes common to children (eg, asthma, respiratory syncytial virus, bronchiolitis, and pneumonitis) can cause lung hyperinflation, resulting in downward displacement of the liver. Other signs of volume overload should also be considered in the evaluation of these patients. If a child with an enlarged liver fails to respond to the initial fluid administration, radiological examination of the chest may help to evaluate heart size. An abnormal cardiac silhouette may provide radiographic evidence of a cardiac etiology to the child's symptoms.

Crackles may occur late in children who are developing congestive failure, and a gallop may be difficult to discern in infants owing to their rapid heart rate. (See **Chapter 6** for a more extensive discussion.)

1. Hypovolemic Shock

Case Study

A 6-year-old developed profuse bloody diarrhea a few days after returning from a family vacation to Southeast Asia. For the last 3 days, she has had over 10 daily bowel movements, which at first were watery but now are bloody. She has had intermittent abdominal cramping and occasional nonbilious vomiting. Her parents are concerned because she no longer drinks fluids and is lethargic. On physical exam, she is supine and does not respond to simple questions. She has a reduced response to nail bed pressure pain. Her heart rate is 160 beats/min, capillary refill is prolonged at 4 or 5 seconds, and she has a respiratory rate of 20 breaths/min.

Detection

– What is the etiology of shock in this scenario?

– What is her PEWS score?

– Does her travel history to Southeast Asia suggest potential pathogens for bloody diarrhea (eg, *Campylobacter*)?

Intervention

– Would isotonic (eg, normal saline) or hypotonic (eg, 5% dextrose in water) IV fluid be best?

– Would a trial of oral rehydration therapy be appropriate in this context?

Reassessment

– If large volumes of warmed fluid are needed rapidly, would a rapid infuser be the best way to administer them?

Effective Communication

– What type of access is needed? Peripheral and/or central? Intravenous? Intraosseous? Facilitated subcutaneous hydration using recombinant human hyaluronidase?

Teamwork

– How long and how many times should team members try to obtain IV access before attempting intraosseous access? (**Appendix 2**)

This patient's PEWS score is: behavior lethargic = 3, cardiovascular tachycardia of 160 beats/min (which is >30 above the normal rate of 70 to 110 beats/min for 6-year-old children) = 3, respiratory rate within normal limits = 0, for a total = 6. The most common cause of shock in the pediatric patient is acute hypovolemia resulting from increased fluid and electrolyte losses (gastrointestinal disorders) or blood loss resulting from severe trauma. Globally, approximately 2 billion gastroenteritis cases occur each year. Acute gastroenteritis is the second leading cause of death in children younger than 5 years, with over 2 million deaths worldwide.

A detailed medical history, including travel history, should be obtained from the child's parent or caregiver and referring institution. A history of increased fluid losses (vomiting and diarrhea), lethargy, and decreased urine output is found in children with hypovolemic shock. Blood pressure is maintained longer in hypovolemic children than in adults; thus, blood pressure is not a clear indicator of volume and perfusion status. Tachycardia, mental status, capillary refill, decreased urine output, and temperature of extremities are much more reliable indicators of hypovolemia in children because they may become abnormal much earlier than hypotension. The primary treatment is appropriate intravascular fluid volume resuscitation with isotonic solutions (**Chapter 6** and **8**). In resource-limited environments, oral rehydration therapy using oral rehydration salts, as recommended by the World Health Organization, has been shown to be effective, especially in cases of diarrhea caused by *Vibrio cholerae*. Facilitated subcutaneous hydration is an effective way of treating mild to moderate dehydration, especially if oral hydration has not been successful and/or if IV access would be difficult. A small-gauge needle (eg, 22-24 gauge butterfly needle) is inserted into the subcutaneous space on the thigh, abdomen, back, or arm of the child. Recombinant human hyaluronidase is injected and catalyzes the hydrolysis of hyaluronan in the extracellular matrix, thereby decreasing its viscosity and increasing tissue permeability. This allows for the painless flow of the isotonic fluid (normal saline or lactated Ringer solution) into the subcutaneous space. The typical bolus is 20 mL/kg with a maximum of 500 mL to run over 1 hour; this can be repeatedly used to assist in rehydration for up to 72 hours or until IV access is obtained.

2. Cardiogenic Shock

Case Study

A 3-year-old boy of Asian ancestry was diagnosed with Kawasaki disease 5 weeks ago. His parents brought him in after 4 days of a high fever (>102.2°F [39°C]), extremely red eyes without thick discharge, diffuse truncal rash, red skin on the palms of his hands, and swollen neck lymph nodes. These have all resolved, but now the boy is complaining that his chest hurts. His parents note that he was much more active last week than this week and that he seems swollen, especially in his ankles and feet. On physical exam, he is pale, sleeping, and breathing fast (55 breaths/min). His feet have pitting edema, his capillary refill is 3 seconds, and his liver is enlarged.

Detection

– What is his PEWS score?

– What studies should you perform to confirm your diagnosis?

Intervention

– What medications and/or procedures are required?

Reassessment

– How will you measure this child's response?

Effective Communication

– What should the team be accomplishing first?

Teamwork

– What other specialists should be involved?

This patient's PEWS score is: behavior sleeping = 1, cardiovascular pale color and capillary refill 3 seconds = 1, tachypnea of 55 breaths/min (which is >10 above the normal range of 35 to 40 breaths/min) = 1, for a total = 3. Congestive heart failure in children is most often due to underlying congenital heart anomalies and often precedes cardiogenic shock. This can often be the result of acute or chronic changes in the heart's preload, afterload, contractility, rate, and/or rhythm. In the case of congenital heart disease, signs and symptoms vary depending on the type of lesion (**Chapter 16**). Myocarditis should be considered in all children with new-onset congestive heart failure. The usual symptoms of heart failure are fatigue, dyspnea on exertion, poor feeding in infants, acute onset edema, and fever. In this patient, there is also an antecedent history of Kawa-saki disease, in which myocarditis and/or ischemia are known complications that can present with heart failure. Early transfer to a pediatric ICU for further monitoring, inotropic support, and a complete evaluation by a pediatric cardiologist is recommended. Other common etiologies of cardiogenic shock include hypoxic-ischemic episodes after acute life-threatening events, near drowning, or strangulation and post-infectious myocarditis.

3. Distributive Shock

Case Study

A 12-year-old boy with a known allergy to Hymenoptera (eg, bees, wasps, hornets) stings is stung by a bee while playing outside. The stinger is still embedded in his skin. His father uses a credit card to scrape across the stinger and successfully removes it. Soon afterwards, the boy develops severe itching and hives all over his body and feels light-headed. His father rushes him to the emergency department where he is now complaining of trouble breathing, nausea, and facial swelling. He is irritable, warm, and tachycardic (heart rate of 128 beats/min); he exhibits bounding pulses and capillary refill of 4 seconds. His respiratory rate is 35 breaths/min.

Detection

– What is his PEWS score?

– What is the etiology of shock in this scenario?

Intervention

– What would be your immediate response?

– What medications does he need?

Reassessment

– Does his airway need to be secured?

– Is his breathing improving?

Effective Communication

– Who needs to know of his anaphylactic reaction? Family? School?

– How would you help the family prevent this reaction in the future? Medical alert bracelet? Epinephrine auto-injector?

Teamwork

– How can the team quickly evaluate and treat the boy's circulatory, airway, and respiratory compromise?

This boy's PEWS score is: behavior irritable = 2, cardiovascular capillary refill of 4 seconds = 2, respiratory rate of 35 breaths/min (which is >10 breaths/min above the normal rate of 19 to 21 breaths/min) = 1, for a total = 5. Distributive shock occurs as a result of an abnormality of vasomotor tone. This has the effect of redistributing blood volume and flow to the periphery, causing relative hypovolemia. In essence, the intravascular space has increased size without a corresponding increase in intravascular volume. Patients appear flushed and have warm extremities with bounding pulses. They are typically tachycardic with a wide pulse pressure and brisk capillary refill. In anaphylaxis, histamine is released from the mast cells and basophils in the lung and gastrointestinal tract. The signs of anaphylaxis include wheezing, respiratory distress, urticaria, vomiting, swelling of the face and tongue, and hypotension. Neurogenic shock is accompanied by a loss of sympathetic tone, which results in decreased peripheral vasomotor tone, vasodilatation, and a lack of compensatory rise in heart rate in response to shock. Neurogenic shock is resistant to fluid resuscitation but is responsive to appropriate α-adrenergic agents (eg, phenylephrine, 0.1-0.5 μg/kg/min).

Anaphylaxis is an acute, potentially life-threatening syndrome, with multisystem manifestations resulting from the rapid release of inflammatory mediators. Foods – most commonly, milk, eggs, wheat, and soy – can be a significant trigger for immunoglobulin E-mediated anaphylaxis in children. Peanuts and fish are among the most potent triggers, but other common stimuli include preservatives in food and drugs, medications (antibiotics), insect venom (bee stings), and bioactive substances (blood products).

Case Study

A 14-year-old IV drug user had been complaining of redness over the vein she injected and left arm

swelling and pain beginning 2 days after injecting heroin. Now she is obtunded, confused, and appears gray and mottled. She is poorly responsive, tachycardic (heart rate of 130 beats/min), tachypneic (respiratory rate of 45 breaths/min), and hypotensive (blood pressure 70/40 mm Hg and mean arterial pressure of 50 mm Hg). Blood culture revealed Gram-positive cocci in clusters consistent with *Staphylococcus aureus*.

Detection

– What type of shock does this patient have?

Intervention

– What monitoring, IV access, and medications does this patient need?

Reassessment

– After starting vasoactive medications, how does one measure effectiveness?

Effective Communication

– How should the life-threatening nature of this illness be communicated to all her caregivers?

Teamwork

– How can the healthcare providers work together to obtain the best possible clinical outcome for this girl and her family?

As in adults, the most common cause of distributive shock in pediatric patients is sepsis. Septic shock is characterized by changes in mental status, fever or hypothermia, and perfusion abnormalities, such as vasodilation ("warm shock") early in septic shock followed by vasoconstriction ("cold shock").

The clinical presentation of warm shock is vasodilation, tachycardia, bounding pulses, brisk capillary refill, and widened pulse pressure. These patients have low systemic vascular resistance and high cardiac output. Cold shock reflects a low cardiac output state with poor peripheral perfusion and high systemic vascular resistance. These individuals will present with tachycardia, prolonged capillary refill, mottled skin, and weak pulses. The therapeutic goal in septic shock is to restore and maintain optimal organ perfusion and oxygenation as evidenced by normal mentation and adequate urine output (≥1 mL/kg/h). Children in shock are usually severely hypovolemic and will respond to aggressive fluid resuscitation (**Chapter 6**). Isotonic fluid boluses in 20 mL/kg increments are usually helpful. Crystalloids (eg, normal saline or Ringer lactate solution) or colloids (5% albumin or dextran) may be used. Typical fluid requirements range from 40 to 200 mL/kg during the initial phase of resuscitation.

Tachycardia is a normal response to any stress in infants and children.

After fluid resuscitation, the first choice of vasopressor support in warm shock is norepinephrine (noradrenaline) [0.05-0.3 μg/kg/min], then epinephrine (adrenaline) [0.05-0.3 μg/kg/min]. Dopamine (5-10 μg/kg/min) is recommended as the first choice in patients with fluid-refractory cold shock, followed by epinephrine if unresponsive to dopamine. Dobutamine, a β_1-agonist, may be given to patients with low cardiac output and elevated systemic vascular resistance states (vasoconstricted) after fluid resuscitation. These continuous IV medications are best delivered through a central access line to avoid extravasation injury from a peripherally inserted catheter. Use of corticosteroids (hydrocortisone, 1-2 mg/kg/day) is indicated in patients with vasopressor-resistant shock, purpura fulminans, or suspected adrenal insufficiency (chronic corticosteroid use, malignant disease, collagen vascular disorders, etomidate for intubation). The pathophysiology of septic shock is complex and exhibits the features of hypovolemic, distributive, and cardiogenic shock. The therapeutic goal in septic shock is to expeditiously restore and maintain optimal organ perfusion.

In cases of shock, it is desirable to maintain an optimal hemoglobin concentration for the patient's age (minimum 10 g/dL).

4. Obstructive Shock

Case Study

A full-term 10-day-old male neonate who had been breastfeeding well for the first 7 days of life is no longer gaining weight, feeds poorly, has only one wet diaper each day, and started sleeping through the night for the last 3 days, according to his parents. On physical exam, he is pale, listless, and cyanotic. He is breathing fast with a palpable liver 5 cm below the costal margin. He has a harsh sounding heart murmur, poor capillary refill, and no femoral pulses.

Detection

– What is his PEWS score?

– What studies should you perform to confirm your diagnosis?

Intervention

– What medications and/or procedures are required?

– What are the side effects of prostaglandin E_1 (PGE_1; alprostadil) therapy?

Reassessment

– How will you measure the neonate's response?

– How will you prevent adverse complications (ie, fever, flushing, apnea) as a result of PGE_1 therapy?

Effective Communication

– What should the team be accomplishing first?

Teamwork

– What other specialists should be involved?

The patient's PEWS score is: behavior lethargic = 3, cardiovascular cyanosis = 2, tachypnea = 1, for a total = 6. Patients presenting with obstructive shock will exhibit a rapid and often precipitous deterioration in hemodynamic status. Neonates (up to 28 days) with ductal-dependent lesions (eg, coarctation of the aorta, hypoplastic left heart, aortic stenosis) will typically present in the first few weeks of life in profound shock and have a history of poor feeding, tachypnea, lethargy, cyanosis, thready or absent femoral pulses, and poor or absent urine output (obstructive shock). Prompt initiation of PGE_1 (alprostadil; 0.05 to 0.1 µg/kg/min), inotropes, and isotonic fluids are lifesaving. Administration should not be delayed to await a confirmatory echocardiogram. Common side effects of PGE_1 are fever, flushing, and apnea; the latter may occur without warning and at anytime during therapy. When transferring a patient receiving PGE_1 to another facility, it is strongly recommended to secure the airway with an endotracheal tube. Once the ductus arteriosus has reopened, the amount of PGE_1 can be reduced. Hyperventilation and hyperoxia must be avoided to prevent excessive pulmonary vascular bed dilation, leading to preferential pulmonary blood flow away from the systemic side (aorta), across the now-patent ductus arteriosus, to the pulmonary side (pulmonary artery). This will worsen systemic perfusion and exacerbate systemic shock.

Patients with nonductal-dependent lesions present outside the neonatal period with a history of tachycardia, gallop rhythm, heart murmur, tachypnea, hepatomegaly, and failure to thrive. These patients will often respond to diuresis with a loop diuretic (furosemide, 0.5-1 mg/kg) and to inotropic support (milrinone [amrinone], 0.5-1 µg/kg/min, or dobutamine, 5-10 µg/kg/min) and/or afterload reduction with an angiotensin-converting-enzyme inhibitor rather than fluid resuscitation (**Chapter 16**).

!

Administration of prostaglandin should not be delayed to await a confirmatory echocardiogram.

V. NERVOUS SYSTEM

Case Study

A 15-month-old girl was left with the mother's friend. When she returned, she was told her child had "slept" all day and did not eat anything. When her mom could not wake her, she brought her straight to the hospital. On physical examination, the child is unresponsive to painful stimulation. She has burns to the dorsum of her hands and multiple linear bruises to her back and buttocks. She is breathing deeply and rapidly at 60 breaths/min. She is pink with a capillary refill of 2 seconds. A head computed tomography scan reveals a subdural blood collection on the right parietal area with midline shift to the left. Her right pupil is dilated and not responsive to light. Upon return from radiology, she begins to have seizures.

Detection

– Are the clinical findings consistent with the history provided?

– What are the most life-threatening injuries?

Intervention

– What steps must be taken first?

Reassessment

– Is the girl responding to therapy?

– Does intracranial pressure need to be monitored directly?

Effective Communication

– How should the team interact with the mother, father, and other caregivers?

– What needs to be documented at the time of presentation and by whom?

Teamwork

– What is the role of medical and nursing personnel in cases of suspected abusive trauma?

– When should law enforcement and social services be contacted in suspected cases of abusive trauma?

Any infant or young child with a depressed level of consciousness, seizures, and/or coma should be evaluated for the possibility of abusive trauma (eg, child abuse, shaken baby syndrome) even if there are no outward signs of injury. Because of the pliability of their bones, children can sustain internal injury without significant external bruising or rib fractures. Furthermore, infants can lose a significant amount of blood in the cranium, resulting in hypovolemic shock. In this case, the child is a victim of abuse and has sustained a closed head injury as a result of abusive head trauma.

The Glasgow Coma Scale is frequently used to evaluate a child's neurological status and level of consciousness. This scale is difficult to apply in children, even when it is adapted for age (**Chapter 15, Table 15-1**). When assessing the need for further intervention, careful attention should be paid to the pediatric patient's ability to maintain the airway, pupillary reaction, verbal responses, motor system reaction, and the fontanelle status in infants. The fontanelle, which usually closes by 24 months of age, is best examined when the child is calm and sitting up. If the child is supine and/or crying, the anterior fontanelle may feel full and tense, thereby giving a false impression of increased intracranial pressure.

The pupillary response is used to rapidly evaluate brainstem function, but abnormalities in pupillary response, such as miosis and mydriasis, may also be found in many toxidromes. Other pertinent physical signs include muscle tone, muscle strength, facial symmetry, ataxia, and any abnormal movements of extremities and facial musculature. In infants, seizures may be characterized by decreased alertness (the infant does not regard parents or track an object across the visual field), autonomic changes (tachycardia, elevated blood pressure, and dilated pupils), apnea, cyanosis, and subcortical muscle activity (bicycling movements of the legs, swimming movements of the arms, sucking, or tongue thrusting). Tonic-clonic muscle motion may not occur during seizures in infants because neuronal myelination and connections have not fully developed. The treatment of seizures is covered in depth in **Chapter 15**. If the seizure or the medications used to treat the seizure significantly compromise the patient's airway and breathing, an endotracheal tube should be inserted. Neuromuscular blocking agents do not treat seizures and will obscure their physical manifestations. Neuromuscular blocking agents should only be used to facilitate endotracheal intubation. The pediatric intensivist and pediatric neurologist should be involved early in the care of status epilepticus.

Hyponatremia and hypoglycemia should be considered in infants and small children presenting with coma and/or seizures. A point-of-care glucose test is needed as part of the primary survey to rule out hypoglycemia as a cause of altered mental status. Hypocalcemia will cause seizures in infants and is commonly associated with tetany. Treatment of severe symptomatic hypocalcemia with IV calcium chloride or calcium gluconate through a centrally placed catheter is preferred so as to avoid peripheral subcutaneous tissue extravasation and resultant injury. For treatment of electrolyte abnormalities, see **Chapter 8**.

Hyponatremia, hypoglycemia, and hypocalcemia should be considered in infants and children presenting with seizures or altered mental status.

VI. SEPSIS

Because of their incompletely developed immune systems, children and neonates are treated with empiric antibiotic therapy more frequently than are adults. The positive predictive value of blood cultures in the pediatric population is limited and dependent on the number performed, amount of blood obtained, and pathogen involved. The neonatal population is especially vulnerable, and the following factors increase their risk of infection:

- Decreased polymorphonuclear white cell function and storage reservoir
- Poor ability for antibody synthesis
- Reduced delivery of phagocytes to inflammatory sites
- Passive maternal immunity depleted by 6 months after birth

Most maternal antibodies are transported across the placenta in the third trimester of pregnancy, limiting passive maternal immunity in premature neonates. Adult levels of immunoglobulin are not reached until 4 to 7 years of age.

Fever always suggests the possibility of serious bacterial disease. In the febrile infant younger than 2 months, antibiotics are considered an emergency drug and often begun before a source of infection is identified. In these infants, the most common causes of life-threatening bacterial infections in the United States are group B streptococci (*Streptococcus agalactiae*), *Escherichia coli*, *Listeria monocytogenes*, and *Enterococcus*. Before the age of 3 years, the risk of occult bacteremia is increased if the temperature is >104°F (>40°C) or the white blood cell count is <500 cells/mm³ or >15,000 cells/mm³. Absolute neutrophil count <1,000 cells/mm³ and significant neutrophil bandemia of 25% to 30% are also markers of severe bacterial infection in children. A full workup is recommended: blood culture, urine culture, and lumbar puncture for cerebral spinal fluid culture as clinically indicated. In children aged 2 months to 2 years, *Streptococcus pneumoniae*, *Staphylococcus aureus* (methicillin-sensitive or -resistant), *Haemophilus influenzae*, *Neisseria meningitidis*, and *Salmonella* are the organisms most associated with serious infections. Clinical signs of sepsis may involve respiratory distress, temperature instability (including hypothermia), and gastrointestinal distress.

An exanthema or rash is often seen, and its characteristic color and distribution pattern may be specific to a particular infection. Examples of such exanthemas would be petechiae or purpura suggestive of meningococcemia, vesicular lesions indicative of herpes simplex or Coxsackie viral infections, or erythematous rash with evidence of peeling secondary to staphylococcal or streptococcal infections. Definitions for sepsis and organ dysfunction in pediatric patients are discussed at length in **Chapter 7**.

The features unique to the care of infants and children, as discussed in this chapter, severely limit the

margin for error in the treatment of critically ill or injured pediatric patients. Therefore, specialty consultation should be requested early. Children tend to deteriorate and recover more quickly than their adult counterparts. It is incumbent on those caring for critically ill children to **DIRECT** their care: **detect** a child with a severe injury or illness; **intervene** in a timely manner; **reassess** each intervention with appropriate monitoring and diagnostic tests; **effectively communicate** with each other so that key information is relayed and lifesaving therapies implemented; and practice **teamwork**, for no one person has all the requisite knowledge, training, and capability to care for a severely ill or injured child.

Key Points

Assessment of the Critically Ill Child

- Every healthcare provider performing a general examination should ask, "Does this child look sick?" If the answer is yes, the child likely is critically ill.
- The Pediatric Early Warning System (PEWS) is an evaluation tool utilized as an early indicator of impending clinical deterioration.
- Children should always be examined in the position they spontaneously assume for comfort. Forcing a child into a different position may worsen respiratory distress and even precipitate respiratory arrest.
- Early signs of respiratory distress in children include tachypnea, grunting, and nasal flaring.
- Ensuring a patent airway is the most important initial step in treating a child with respiratory compromise. Chest movement does not ensure a patent airway.
- A child's perfusion status is best assessed initially by mental status, capillary refill, urine output, and temperature of extremities. Hypotension is a late finding in pediatric shock. Timely recognition of the shock state and aggressive intervention are essential to obtaining an optimal outcome.
- Infants and young children presenting with seizures should be evaluated for electrolyte imbalances and hypoglycemia.
- Young infants face an increased risk of infection due to their immature immune systems.
- Empiric antibiotics are considered emergency drugs for febrile infants younger than 2 months.

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AIRWAY MANAGEMENT

Objectives

- Recognize the signs and symptoms of an unstable airway.
- Detect the signs and symptoms of respiratory failure.
- Identify unique anatomic and physiologic variables involved in the management of the pediatric airway.
- Describe how to open and maintain an airway manually and with the help of airway adjuncts.
- Explain how to support a patient with bag-mask ventilation.
- Understand the process and sequence of pediatric intubation.
- Determine the potential for difficult intubation and discuss alternative methods of establishing a stable airway.

Case Study

An 18-month-old child is brought to the hospital with a 1-day history of a harsh barking cough. On physical examination, he has significant chest wall retraction with abdominal breathing. Oxygen saturation is 88% on room air.

Detection

– What is this child's physiologic status?

– What signs and symptoms are indicative of respiratory distress?

– What are the most likely diagnosis and the worst possible diagnoses?

Intervention

– What are the most immediate treatment priorities?

– What initial treatment options may be attempted to maintain the airway?

– If the patient is intubated, what steps must be taken to ensure that the procedure is as safe as

possible?

– What size and type of laryngoscope blade would you select?

– What size of endotracheal tube would you select, and what would be the appropriate depth of tube placement?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– When the patient's clinical status changes, who needs the information and how will it be disseminated?

– Where is the best place to manage the care of this patient?

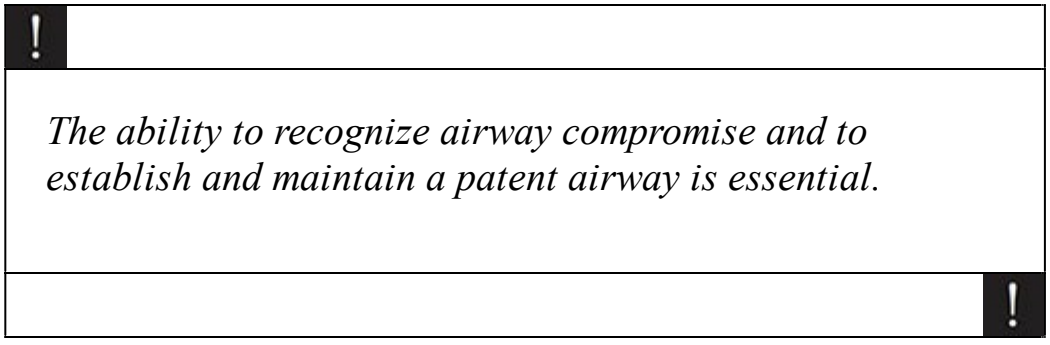
Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

I. INTRODUCTION

Respiratory distress occurs frequently in children. It can develop from problems at all levels of the respiratory tract, as well as the neuromuscular and cardiovascular systems. The majority of cardiopulmonary arrests in children are heralded by respiratory failure. Recognition and interruption of the progression from respiratory compromise to respiratory failure is fundamental to pediatric airway management. The ability to recognize airway compromise and to establish and maintain a patent airway, despite age-related anatomic differences, is essential.

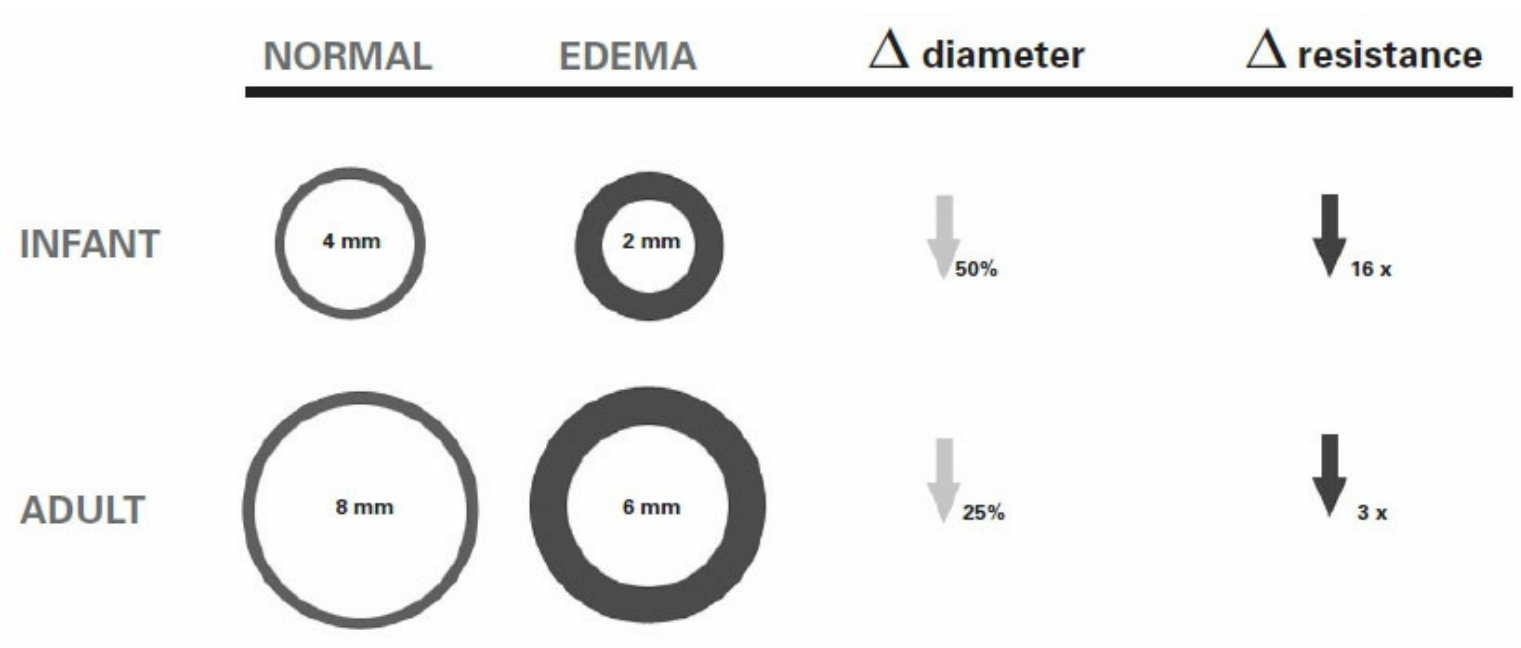


II. ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

The airway of a child changes dramatically from birth to adulthood. To assess and manage airway emergencies, it is necessary to understand certain the anatomic and developmental aspects of a child's airway.

- In children, the nose provides nearly half of the total airway resistance. The infant's nose is short, soft, and small with nearly circular nares. Infants younger than 2 months are obligate nasal breathers. Although the nares double in size from birth to 6 months, they can be easily occluded by edema, secretions, or external pressure. Clearing the nasal passages with suctioning can significantly improve an infant's respirations.
- A child's tongue is large relative to the oral cavity. This disproportion is increased in developmental disorders such as Pierre Robin sequence and severe micrognathia. A large tongue in a relatively small oral cavity increases the difficulty of visualizing the larynx during laryngoscopy. Backward displacement of the tongue into the posterior pharynx because of decreased muscle tone in sleep, depressed level of consciousness from head injury, sedation, or other nervous system dysfunction can result in upper airway obstruction.
- The level of the larynx in the neck changes from C2 in neonates to C3 to C4 in children and ends up at C5 to C6 in adults. In infants, the epiglottis is at the level of the first cervical vertebra and overlaps the soft palate. The high position of the larynx, combined with a large tongue and a small mandible, contributes to an infant's susceptibility to airway obstruction.
- The high position of the larynx also makes the angle between the base of the tongue and the glottic opening more acute, which increases the difficulty of visualizing the vocal cords during laryngoscopy. Therefore, a straight laryngoscope blade, such as a Miller blade, may be superior for creating a straight plane from the mouth to the glottis.
- An infant's epiglottis is long, soft, and omega (Ω)-shaped, whereas an adult's epiglottis is shorter, more rigid, and flatter. A longer and softer epiglottis is more difficult to control during laryngoscopy. Using a straight laryngoscope blade, which directly lifts the epiglottis and exposes the vocal cords, can overcome this difficulty.
- A pediatric larynx is funnel-shaped, with the subglottic portion angled posteriorly to the supraglottic portion. Consequently, the narrowest portion of the larynx is in the subglottic space at the level of the cricoid cartilage. As a result, inflating the cuff of an endotracheal tube could lead to subglottic edema. The adult larynx is barrel-shaped, with the narrowest portion at the level of the vocal cords, and is less prone to this complication.
- The internal diameter of a pediatric trachea is approximately one-third that of an adult, resulting in higher airflow resistance. Resistance to airflow is a function of $1/r^4$, where r is the airway radius. A small decrease in airway diameter (2 × radius) due to edema or secretions causes a far greater increase in resistance in a child than in an adult (**Figure 2-1**).

Figure 2-1. Airway Resistance Proportionate to Airway Radius



- The length of a newborn's trachea is approximately 5 cm, and that of an 18-month-old infant is approximately 7 cm. Given this short length, both right main-stem bronchus intubation and unplanned extubation are common.

!
Clearing the nasal passages with suctioning can significantly improve an infant's respiratory condition.
!

- The chest wall of an infant is easily compressible due to the elasticity of its cartilaginous structures.
  - The use of abdominal muscles leads to a characteristic seesaw, or abdominal, breathing pattern.
  - Intercostal, subcostal, and suprasternal retractions become prominent as airway obstruction or lung disease increases the work of breathing.
  - Respiratory muscle fatigue because of fewer slow twitch muscle fibers compared to adults may lead to decreased respiratory effort as respiratory failure progresses.

!
A pediatric larynx is funnel-shaped; the narrowest portion is the cricoid cartilage. An adult larynx is barrel-shaped; the narrowest portion is at the level of the vocal cords.
!

III. ASSESSING RESPIRATORY STATUS

In children, as in adults, assessment of airway patency and spontaneous respiratory effort is a crucial first step. The clinician must recognize signs of distress as well as look, listen, and feel for diminished or absent air movement.

A. Observe the Child's General Appearance

Give careful attention to:

- Muscle tone and spontaneous and reactive movement
- Alertness and interaction with the environment and with the caregiver
- Inconsolable crying or agitation
- Ability to speak or cry (phonation and crying require air movement)
- Injury to the airway or other conditions (eg, cervical spine fracture or facial burns) that will affect assessment and manipulation of the airway
- Signs of congenital malformations of the face, mouth, or tongue that might contribute to respiratory problems
- Signs of airway obstruction due to a decreased level of consciousness

Ventilation may be adequate with minimal thoracic excursion, but respiratory muscle activity and even vigorous chest movement do not ensure that the tidal volume is adequate.

B. Assess the Child's Respiratory Rate

- The normal respiratory rate varies with age (**Table 2-1** and **Appendix 1**).
- The respiratory rate is best assessed by observation. Expose the child's chest and watch the rise and fall of the chest and abdomen.
- A respiratory rate >60 breaths/min is abnormal in a child of any age.
- An abnormally slow rate may herald respiratory failure.

Table 2-1 AGE-NORMAL RESPIRATORY RATES

Age	Respiratory Rate (breaths/min)
Newborn	30-60
Infant (1-12 months)	30-60

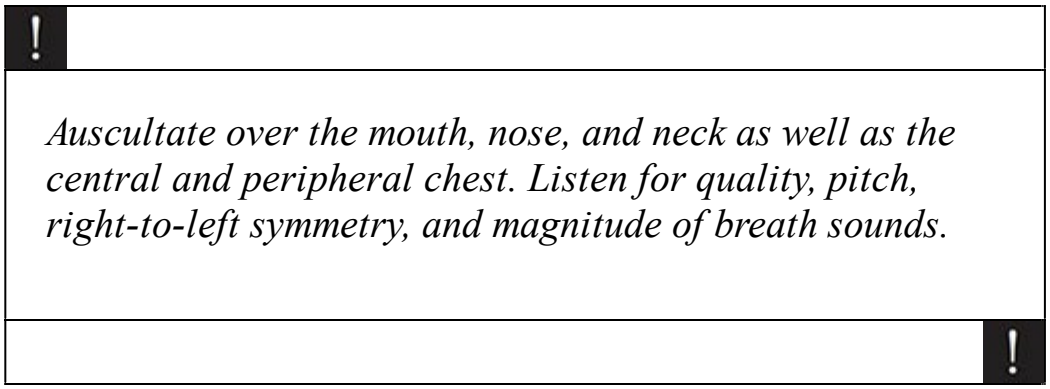
Toddler (1-2 years)	24-40
Preschooler (3-5 years)	22-34
School age (6-12 years)	18-30
Adolescent (13-17 years)	12-16

A respiratory rate >60 breaths/min is abnormal in a child of any age.

C. Consider the Work of Breathing

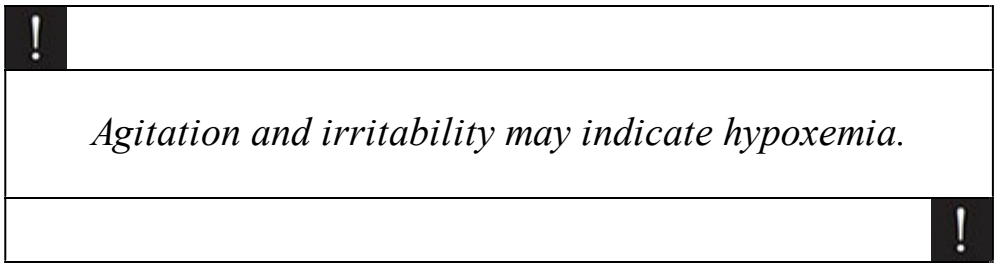
- Intercostal, subcostal, and suprasternal retractions increase with progressive respiratory distress. Decreasing respiratory rate and diminished retractions in a child with a history of distress may signal severe fatigue.
- Increased inspiratory effort often amplifies airway resistance, causing collapse of the compliant upper airway and producing stridor.
- Nasal flaring is an effort to increase airway diameter and is often seen with hypoxemia.
- Grunting is an expiratory noise produced by an effort to prevent airway collapse by generating positive end-expiratory pressure.
- Obstruction or narrowing of the extrathoracic airway (nose, posterior pharynx, larynx, and subglottic space) causes high-pitched inspiratory noise (stridor) and retractions.
- Obstruction or narrowing of the intrathoracic airways results in signs and symptoms that occur predominantly during expiration. Wheezing (high-pitched expiratory sounds) is caused by expiratory obstruction.
- Incomplete obstruction (during which a limited amount of air can be inspired and expired) due to pharyngeal soft-tissue collapse, a mass, or a foreign body in the airway may be associated with snoring, stridor, gurgling, or noisy breathing.
- Complete airway obstruction (during which no air can be inspired or expired) is likely when respiratory effort is visible but breath sounds are absent.
- Crackles are end-inspiratory noises usually heard with parenchymal lung diseases, such as pneumonia and bronchiolitis, and are loudest in the peripheral lung fields.
- Asymmetry of breath sounds is an important clue. Breath sounds may be diminished or absent on

one side in the presence of pneumothorax or pleural effusion or when a tracheal tube is placed in the opposite main bronchus.

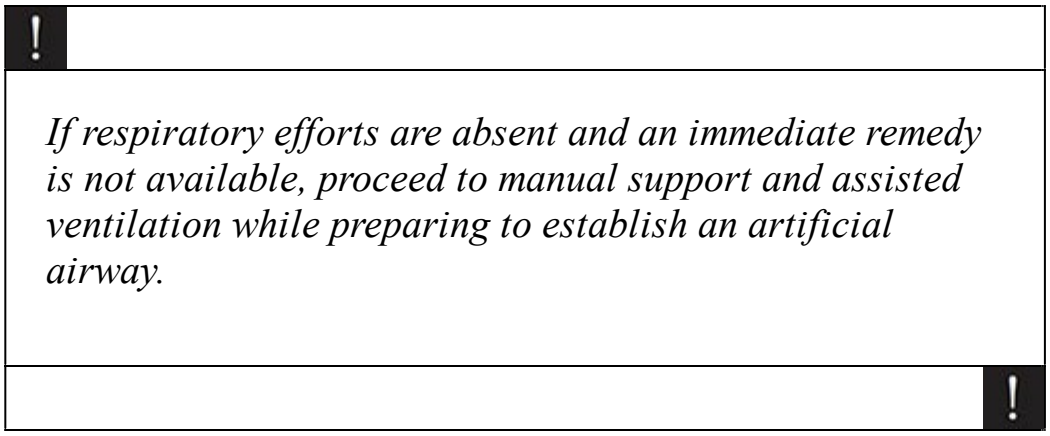

!
Auscultate over the mouth, nose, and neck as well as the central and peripheral chest. Listen for quality, pitch, right-to-left symmetry, and magnitude of breath sounds.
!

D. Evaluate Mental Status

- Agitation and irritability may indicate hypoxemia. Lethargy in the presence of acceptable oxygen saturation may reflect an elevated carbon dioxide tension.



- The presence and strength of protective airway reflexes (ie, cough and gag) must be assessed cautiously. Overly aggressive stimulation of the posterior pharynx while assessing those reflexes may precipitate emesis and aspiration of gastric contents. Moreover, stimulation of the posterior pharynx may convert partial upper airway obstruction (caused by illnesses such as epiglottitis) to complete airway obstruction.


!
If respiratory efforts are absent and an immediate remedy is not available, proceed to manual support and assisted ventilation while preparing to establish an artificial airway.
!

IV. MONITORING RESPIRATORY FUNCTION

A. Longitudinal Physical Assessment

Once a patient is found to be in respiratory distress, close monitoring of respiratory function is essential. Auscultation and observation are important components of this monitoring. Alarm parameters indicating inadequate ventilation and oxygenation should be set appropriately. Constant vigilance is required once a patient is intubated and placed on a mechanical ventilator.

B. Arterial Blood Gas

Arterial blood gas measurement may be helpful when either the etiology or the degree of respiratory insufficiency is unknown. Careful consideration should be given to the fact that arterial puncture is a painful procedure; the resultant agitation might worsen the respiratory distress of some children.

A systematic approach to arterial blood gas interpretation consists of:

- Determining the degree of alveolar ventilation based on the $Paco_2$
- Assessing whether the patient's pH can be explained solely on the basis of $Paco_2$ or if a metabolic component exists
- Determining whether the ventilatory or metabolic event was primary or compensatory
- Assessing the effectiveness of correction of any hypoxemic state

Arterial blood gas disorders are classified into 4 main categories: hypoventilation, alveolar hyperventilation, metabolic acidosis, and metabolic alkalosis.

1. Hypoventilation

Hypoventilation (respiratory acidosis) results in an elevation of Pco_2, which causes the ratio of HCO_3^- to Pco_2 to decrease and results in a decrease in pH. Carbon dioxide retention can be caused by hypoventilation or a ventilation-perfusion inequality. Increasing ventilation will correct respiratory acidosis in most cases. If the patient is unstable, manual bag-mask ventilation is indicated until the underlying problem can be addressed.

If respiratory acidosis persists (as seen in chronic ventilatory failure), the kidneys respond by conserving HCO_3^-. Renal compensation is typically not complete, so the pH approaches—but does not fully return to—7.4.

2. Alveolar Hyperventilation

Alveolar hyperventilation (respiratory alkalosis) results in decreased Pco_2, which in turn elevates the pH. Bicarbonate and base excess remain in the normal range because the kidneys have not had time to establish adequate compensation. Pain and anxiety may cause acute hyperventilation and alkalosis in children. Hypoxemia may lead to increased minute ventilation and respiratory alkalosis.

3. Metabolic Acidosis

A primary decrease in HCO_3^- leads to a decreased pH. Bicarbonate can be lowered by an accumulation of acids in the blood, as in uncontrolled diabetes mellitus, and production of lactic acid in the setting of tissue hypoxia. Respiratory compensation is the result of an increase in ventilation that lowers the Pco_2 and in turn raises pH. Respiratory insufficiency in the presence of metabolic acidosis is concerning due to the degree of acidosis caused by the patient's inability to increase respiratory rate as compensatory response

4. Metabolic Alkalosis

An increase in HCO_3^- raises the ratio of HCO_3^- to Pco_2 and thus the pH. Compensation occurs by hypoventilation and CO_2 retention. Metabolic alkalosis is seen in the child with pyloric stenosis who loses large amounts of stomach acid due to vomiting. The child's ability to compensate for metabolic alkalosis through hypoventilation is limited by hypoxia.

For additional information, please see **Appendix 3**.

C. Pulse Oximetry

Pulse oximetry provides continuous, noninvasive measurement of arterial oxyhemoglobin saturation (Sao_2). **Figure 2-2** shows the oxyhemoglobin saturation curve, which is a graph of Sao_2 versus Pao_2. Under normal conditions, hemoglobin is 90% saturated with oxygen at a Pao_2 of about 60 mm Hg (8 kPa). Raising the Pao_2 above 60 mm Hg does not result in an appreciable increase in hemoglobin saturation. However, due to the sigmoidal shape of the oxyhemoglobin saturation curve, once the Pao_2 drops below 60 mm Hg, the saturation changes rapidly with small changes in Pao_2.

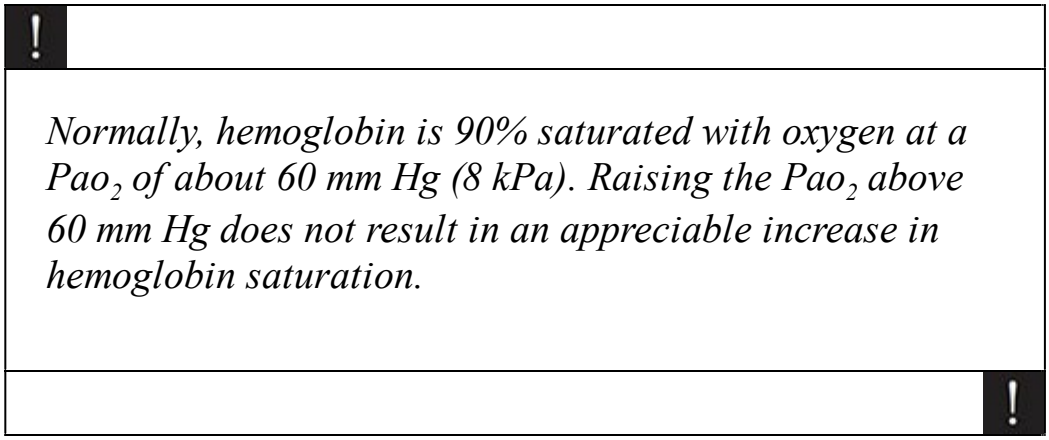
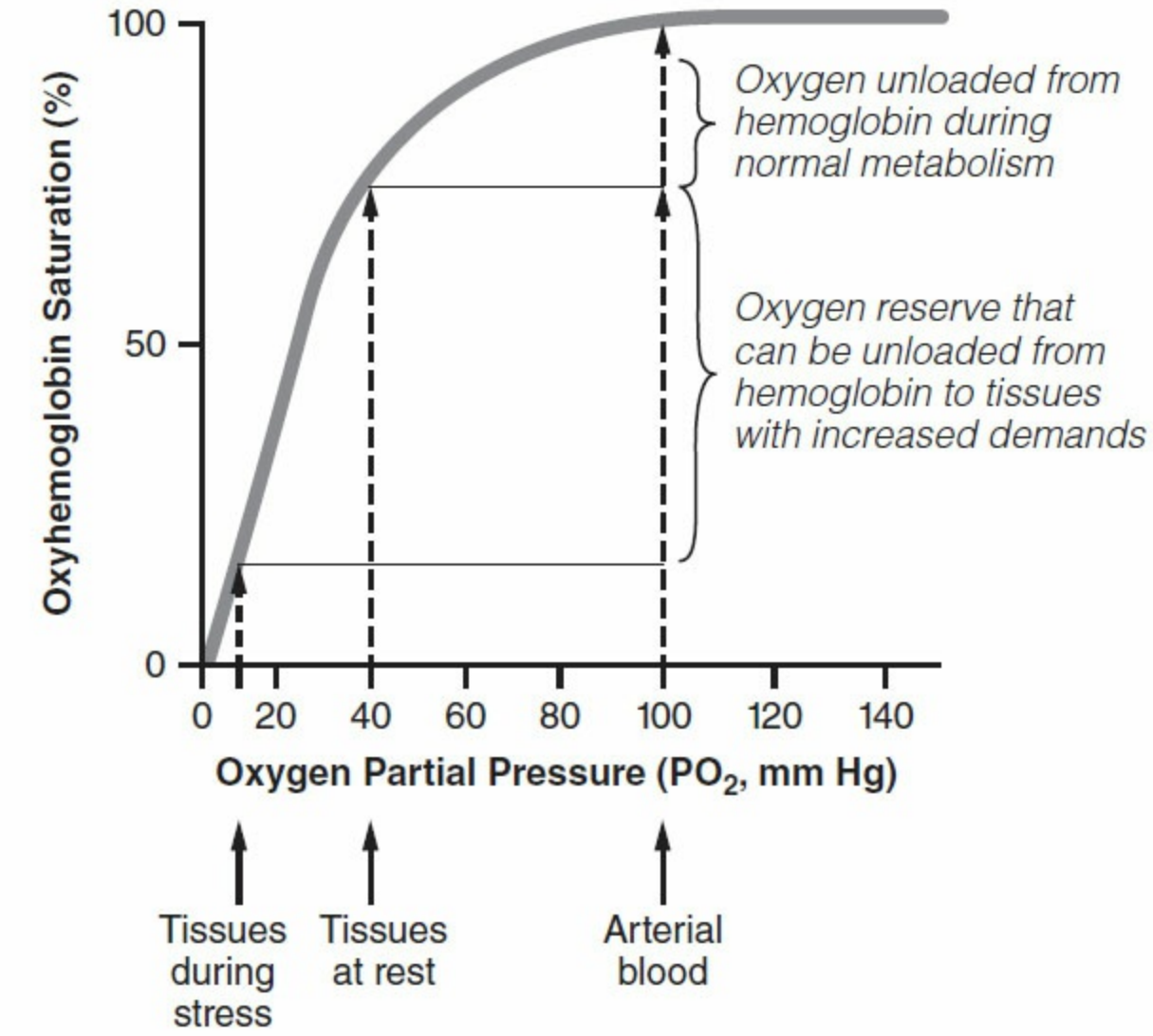

!
Normally, hemoglobin is 90% saturated with oxygen at a Pao2 of about 60 mm Hg (8 kPa). Raising the Pao2 above 60 mm Hg does not result in an appreciable increase in hemoglobin saturation.
!

Figure 2-2. Oxyhemoglobin Saturation Curve


100
50
0
Oxyhemoglobin Saturation (%)
Oxygen unloaded from hemoglobin during normal metabolism
Oxygen reserve that can be unloaded from hemoglobin to tissues with increased demands
0 20 40 60 80 100 120 140
Oxygen Partial Pressure (PO_2, mm Hg)
Tissues during stress
Tissues at rest
Arterial blood

The oxyhemoglobin dissociation curve relates Po_2 to oxyhemoglobin saturation. Near-maximal saturation of hemoglobin occurs at a Pao_2 of 60 mm Hg (8 kPa). Po_2 values above this point provide only a modest increase in oxyhemoglobin saturation. Note, however, that a rapid decrease in oxyhemoglobin saturation occurs when the Po_2 drops below 60 mm Hg (8 kPa).

The limitations to pulse oximetry in managing critically ill patients include the following:

- Pulse oximetry requires pulsatile blood flow. In the presence of shock and poor perfusion, the accuracy and signal strength become unreliable.
- In carbon monoxide poisoning, most pulse oximeters will read falsely high for oxyhemoglobin saturation because carboxyhemoglobin absorbs light at a similar wavelength as oxyhemoglobin. Blood gas measurement with co-oximetry is the only way to determine the true oxyhemoglobin and carboxyhemoglobin saturations.
- Pulse oximetry is not accurate in methemoglobinemia.
- Oxygen saturation may be underestimated in patients with sickle cell disease and acute vaso-occlusive crisis.
- IV dyes and certain colors of nail polish falsely lower pulse oximetry readings.

- The accuracy of the pulse oximeter should be questioned when a patient's appearance and heart rate do not correlate with the pulse oximeter reading.
- As a safety net, it is always prudent to question elevated saturations and accept decreased saturations as accurate until proven otherwise.
- Very few specific saturation monitoring devices are calibrated for children with oxygen saturation on pulse oximetry <90%; others extrapolate data from healthy adults and lose accuracy under around 80%.

D. Exhaled Carbon Dioxide

End-tidal CO_2 monitoring can be used to estimate the arterial CO_2 tension. Quantitative continuous capnography can be performed via nasal cannula or with devices that attach to the tracheal tube. Qualitative (colorimetric) devices that change color when exhaled CO_2 is present are generally used for confirmation of tracheal tube placement and are discussed later in this chapter.

In the presence of low or absent cardiac output, ventilation-perfusion mismatch, airway obstruction, significant rebreathing or tracheal tube leak, end-tidal CO_2 values may not accurately reflect true arterial CO_2 levels.

V. AIRWAY MANAGEMENT

A. Oxygen Delivery Systems

Oxygen should be administered immediately in virtually every setting where respiratory difficulty is suspected. Infants and children consume 2 to 3 times more oxygen per kilogram of body weight than adults under basal conditions and even more when they are ill or stressed. Several key points regarding administration of oxygen to children are as follows

- When administering oxygen to an alert child, the need to increase oxygen delivery must be balanced against the potential for agitation, which increases oxygen consumption.
- If a child is intolerant of one method of oxygen delivery (eg, nasal cannula), another method should be tried (eg, face tent). It is often helpful to have a parent hold the child and assist with placement of oxygen delivery devices (**Appendix 4**).
- Children will often assume a position that maximizes airway patency and minimizes respiratory effort. They should be allowed to remain in their position of comfort.
- Oxygen administration will have limited effect in the presence of airway obstruction. In a somnolent or obtunded child, the airway can be obstructed due to neck flexion, relaxation of the jaw, posterior displacement of the tongue against the pharynx, and collapse of the hypopharynx. Before using airway adjuncts, attempt to open the airway using manual methods. The airway

should be cleared of secretions to optimize the patient's effort.

- Patients who are not moving an adequate amount of air because of poor or weak respiratory efforts should be assisted with a bag-mask device. They may then be transitioned to noninvasive or invasive forms of ventilation. See **Appendix 5** for additional information on airway adjuncts.

!
Oxygen should be administered when respiratory difficulty is suspected.
!

1. Simple Oxygen Mask

The simple oxygen mask is a device that delivers oxygen with a flow rate of 6 to 10 L/min. The oxygen concentration delivered to the patient can reach a maximum of 60% due to entrainment of room air through the exhalation ports of the mask. An oxygen flow rate of at least 6 L/min should be maintained to sustain an optimal inspired oxygen concentration and prevent rebreathing of exhaled carbon dioxide.

2. Partial Rebreathing Mask

A partial rebreathing mask consists of a simple face mask with a reservoir bag. It provides oxygen concentrations of 50% to 60%. During inspiration, the patient draws gas predominantly from the fresh oxygen inflow and the reservoir bag, so entrainment of room air through the exhalation ports is minimized. An oxygen flow rate of 10 to 12 L/min is usually required.

3. Nonrebreathing Mask

A nonrebreathing mask consists of a face mask and reservoir bag with a valve incorporated into one or both exhalation ports to prevent entrainment of room air during inspiration. Another valve between the reservoir bag and the mask prevents flow of exhaled gas into the reservoir. An inspired concentration of 95% can be achieved with an oxygen flow rate of 10 to 15 L/min and use of a well-sealed mask.

4. Face Tent

A face tent is a high-flow soft plastic bucket that children often tolerate better than a face mask. Even with a high oxygen flow rate, inspired concentrations over 40% cannot be reliably provided. A face tent permits access to the face without interrupting oxygen flow. This device is no longer frequently used.

5. Nasal Cannula

A nasal cannula is a low-flow oxygen delivery system that is useful when low levels of supplemental

oxygen are required. The net fraction of inspired oxygen depends on the child's respiratory effort, size, and minute ventilation in comparison with the nasal cannula flow.

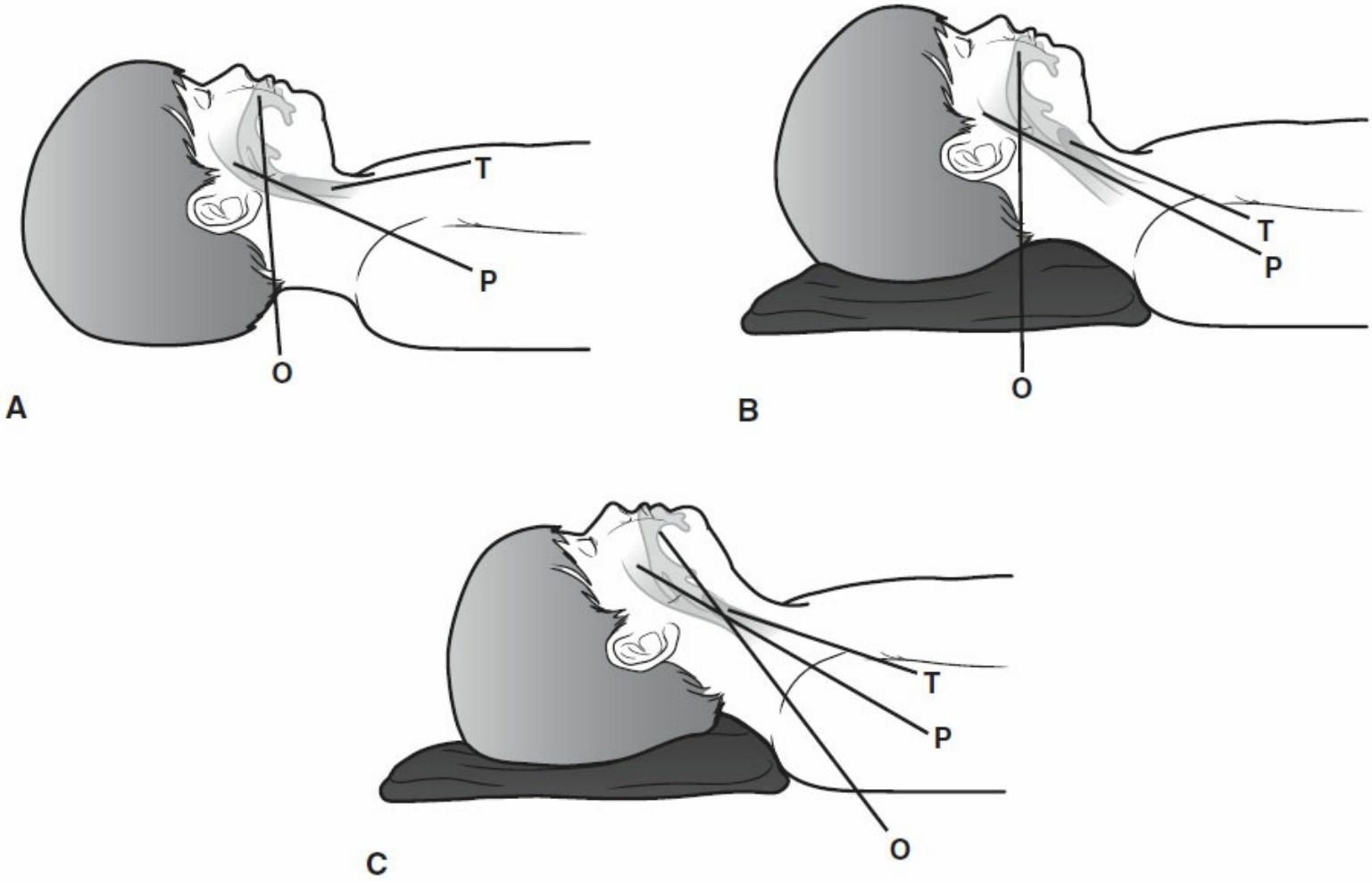
6. High-Flow Nasal Cannula

High-flow nasal cannula (HFNC) is an oxygen delivery system that provides compressed humidified air blended with varying percentage oxygen at a higher flow rate than the simple nasal cannula. A flow rate of up to 10 to 15 L/min is acceptable in pediatric patients but can go up to 60 L/min in adult patients. This helps to eliminate nasopharyngeal dead space, reduce the work of breathing, improve pulmonary compliance, and promote better gas exchange. It is recommended to use 2 L/kg/min for the first 10 kg of body weight, then 0.5 L/kg/min thereafter to an arbitrary maximum of 50 L/min.

B. Maneuvers to Open and Maintain the Airway

The airway has oral, pharyngeal, and tracheal components. Maximal opening of the airway is obtained by aligning all axes of these components, as shown in **Figure 2-3**. Children younger than 2 years have relatively large heads for their body size, which results in slight neck flexion when they are in the supine position. These children usually benefit from a small roll placed under the shoulders to allow the head to fall back into the proper position; a thin towel or blanket is usually sufficient for this purpose. Care must be taken to avoid overextension of the neck, which will result in obstruction. Smaller rolls of towels on either side of the head also prevent the head from moving from side to side. In older children, a small roll under the back of the head helps facilitate proper alignment of the axes.

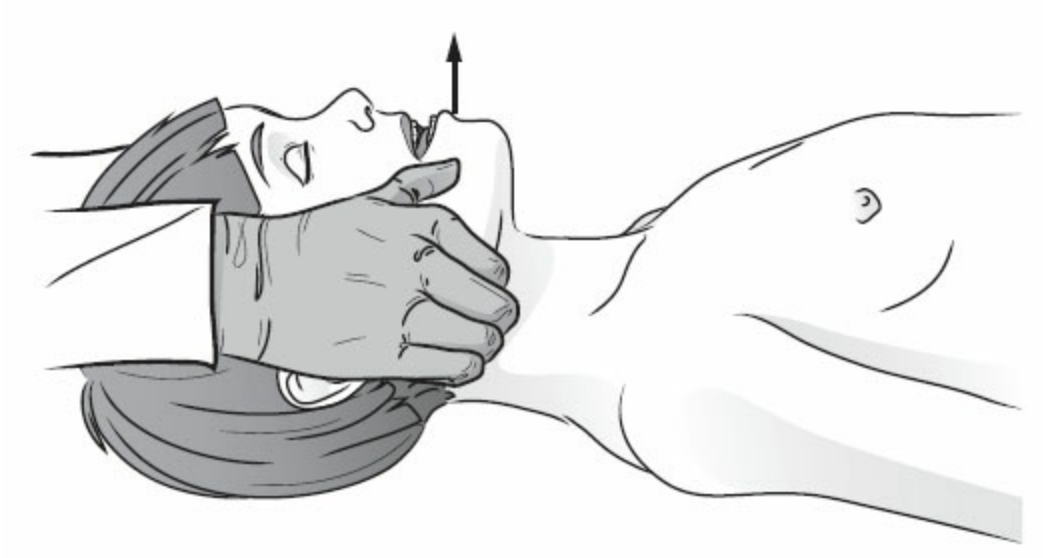
Figure 2-3. Aligning the Oral, Pharyngeal, and Tracheal Axes



A, Maximal opening and visualization of the airway is obtained by aligning the oral (O), pharyngeal (P), and tracheal (T) axes. This is accomplished by placing a patient in the sniffing position. **B**, A folded sheet or towel placed under the occiput aligns the pharyngeal and tracheal axes. **C**, Extension of the neck into the sniffing position then results in approximate alignment of all 3 axes. Proper positioning places the external ear canal anterior to the shoulder. In children younger than 2 years, a folded towel or sheet is placed under the shoulder rather than under the occiput due to the relatively large forehead-to-occiput distance in this age group.

Partial or complete airway obstruction caused by pharyngeal soft tissue collapse or posterior movement of the tongue can usually be relieved by manual maneuvers, including positioning the patient to maximize airway patency and manipulating the jaw and head to open the airway. The first and simplest intervention is the jaw-thrust maneuver (**Figure 2-4**). The clinician's fingers are placed along the posterior rami of the mandible and the mandible is lifted upward or forward. The jaw-thrust maneuver can be performed safely in a patient with a suspected cervical spine injury because neck alignment is not altered. The jaw-thrust maneuver may cause discomfort in the awake patient.

Figure 2-4. Jaw-Thrust Maneuver



Place fingers on the posterior rami of both mandibles and lift in an anterior direction.

A complementary maneuver for opening an obstructed airway is the head-tilt/chin-lift maneuver. The clinician slightly hyperextends the head to place the patient in the sniffing position. In this position, the ear canal is just above or even with the top of the shoulder when viewed from the side.

Jaw-thrust and chin-lift maneuvers ease obstruction in spontaneously breathing patients but should also be used to provide an unobstructed airway when supporting a patient with bag-mask ventilation.

!

Airway obstruction caused by pharyngeal soft tissue collapse or posterior movement of the tongue can usually be relieved by a jaw-thrust or chin-lift maneuver.

!

When a cervical spine injury is suspected, the patient's head should remain in the neutral position at all times, using active stabilization if necessary. Lateral rotation of the head must be avoided. The potential or actual presence of cervical spine injury should not prevent efforts to maintain an unobstructed airway.

C. Oropharyngeal and Nasopharyngeal Airways

An oropharyngeal (OP) airway consists of a flange, a short bite-block segment, and a curved body, usually made of plastic and shaped to provide an air channel and a suction conduit through the mouth. The curve of the OP airway is designed to fit over the back of the tongue to hold it and the soft hypopharyngeal structures away from the posterior wall of the pharynx. The plastic flange should rest against the outer surface of the teeth while the distal end curves around the base of the tongue. If the OP airway is too small, it may push the tongue back over the glottis opening; if it is too large, it may stimulate gagging and emesis or push the epiglottis down, further obstructing the airway.

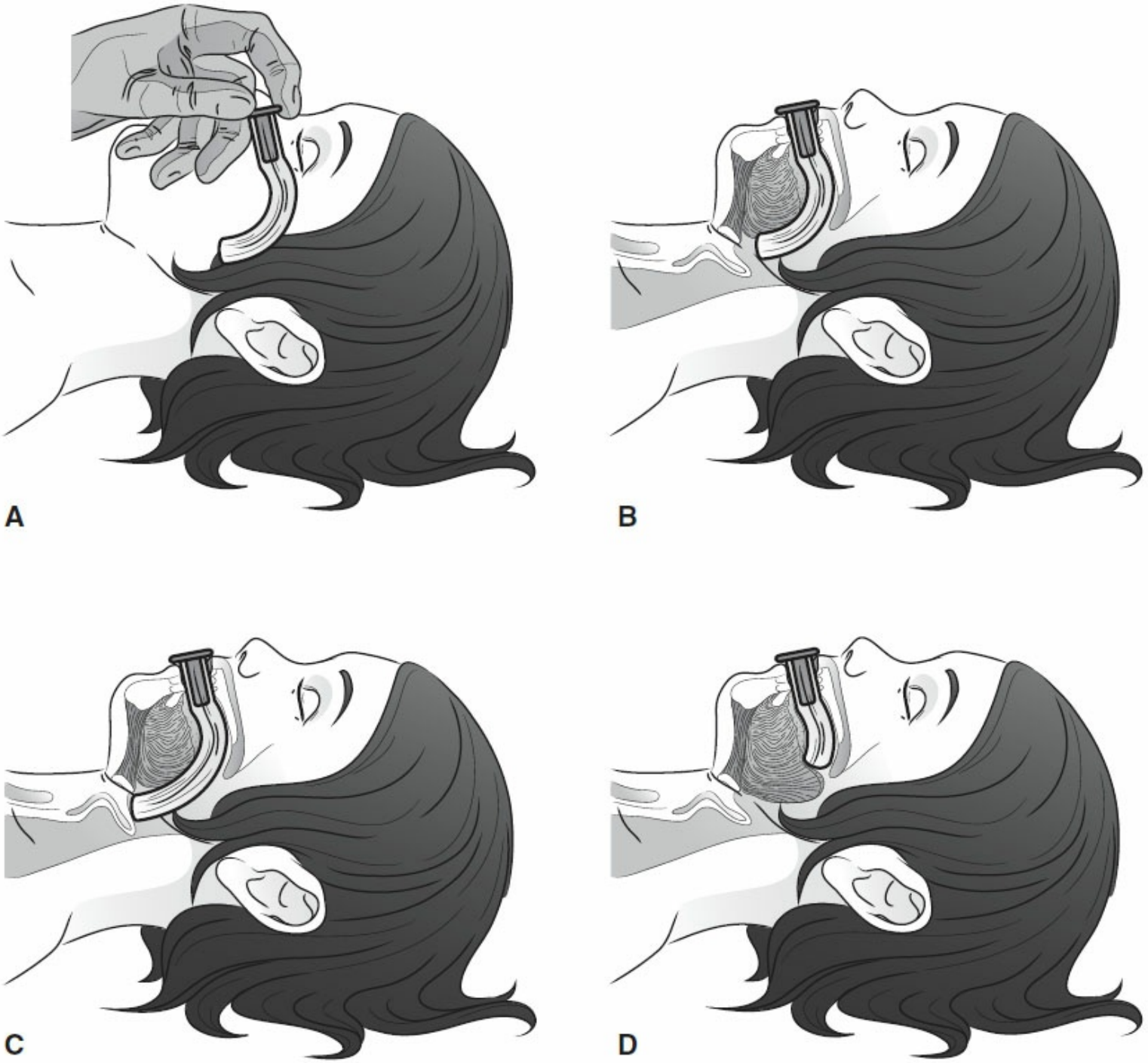
An OP airway can be used to maintain an open airway during bag-mask ventilation, but it is used only in unconscious patients. In conscious or semiconscious patients, it will stimulate gagging, vomiting, and possibly laryngospasm. The technique for inserting an OP airway is given in **Table 2-2** and illustrated in **Figure 2-5**.

Table 2-2	INSERTION OF AN OROPHARYNGEAL AIRWAY

- Select the proper size airway. The correct size extends from the corner of the mouth to the earlobe.
- If the airway is too short, it will not hold the tongue out of the way.
- If it is too long, it may obstruct breathing.
  – Open the patient's mouth.
  – Insert the airway upside down so the tongue does not obstruct the airway.
  – Advance the airway gently until resistance is encountered.
  – Turn the airway 180° so that it comes to rest with the flange on the patient's teeth.

Note: An alternative method, preferable in small infants, is to press the tongue down and forward with a tongue depressor. The oral airway can then be passed, in its final alignment, into position over the tongue. This technique is recommended to avoid palatal trauma and bleeding.

Figure 2-5. Oral Airway Placement



An airway of proper size will relieve obstruction caused by the tongue without damaging laryngeal structures. **A**, the appropriate size can be estimated by holding the airway next to the child's face. The tip of the airway should end just cephalad to the angle of the mandible. **B**, Example of an oropharyngeal airway in correct position and its relationship to airway structures. **C**, If the airway is too large, its tip will hit the epiglottis, pushing it downward and obstructing the glottic opening. **D**, If the airway is too small, it will worsen obstruction by pushing the tongue backwards into the posterior pharynx.

A nasopharyngeal (NP) airway is a soft rubber or plastic tube that provides a conduit for airflow between the nares and the pharynx. It pushes the tongue forward, which relieves airway obstruction. The diameter of an NP airway should be the largest that will easily pass through the nostril, and its length should extend to the nasopharynx, but not be so long that it obstructs gas flow through the mouth or touches the epiglottis.

NP airways are less likely to stimulate vomiting and may be used in patients who are responsive but need assistance in keeping the tongue from obstructing the airway. These devices are especially

helpful in the presence of facial and airway deformities such as those seen in Pierre Robin syndrome.

Relative contraindications to the NP airways include coagulopathy, basilar skull fracture, and nasal infection or deformity. Unlike OP airways, they may be used in conscious patients. The technique for placing an NP airway is outlined in **Table 2-3** and illustrated in **Figure 2-6**.

Table 2-3	INSERTION OF A NASOPHARYNGEAL AIRWAY

- Choose an airway of the proper size. The length of the airway should be roughly the distance from the tip of the nose to the meatus of the ear.
- Lubricate the airway with surgical lubricant.
- Alleviate vasoconstriction with phenylephrine nose drops (and topical anesthesia with lidocaine if the patient is awake) if possible before any nasal instrumentation. In an acute situation, the lubricating qualities of lidocaine ointment may suffice.
- Insert the tip of the airway perpendicular to the face, not upward toward the cribriform plate. The bevel should point toward the base of the nares or toward the septum. If the airway cannot be inserted into one nostril, try the other.
- Listen by ear or stethoscope for air movement through the airway as it is placed. It is correctly positioned when airflow through the tube is present.
- Secure the airway, then attend to maintaining its patency and position. A nasopharyngeal airway should be treated like an endotracheal tube with regard to suctioning and patency. If airflow cannot be auscultated, the tube is either obstructed or malpositioned.

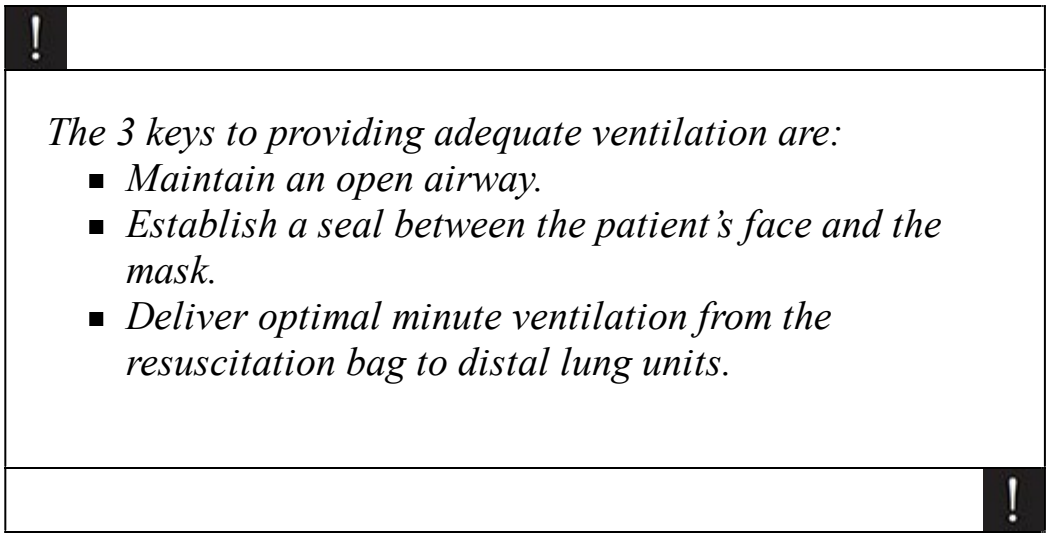
Note: If a commercial nasopharyngeal airway of the proper size is not available, an endotracheal tube of appropriate size can be used instead. Measure the distance from the tip of the patient's nose to the meatus of the ear and use that measurement to determine how much extra length should be cut from the tube before insertion.

Figure 2-6. Endotracheal Tube Used as a Nasopharyngeal Airway

Example of an endotracheal tube used as a nasopharyngeal airway. The insertion technique is described in **Table 2-3**. Note that the 15-mm adapter must be inserted firmly and that the tube must be taped securely to prevent inadvertent advancement.

D. Bag-Mask Ventilation

Bag-mask ventilation is used to help apneic patients and those with inadequate spontaneous ventilation. Knowing how to provide adequate bag-mask ventilation is the most important airway management skill that any healthcare provider can possess.



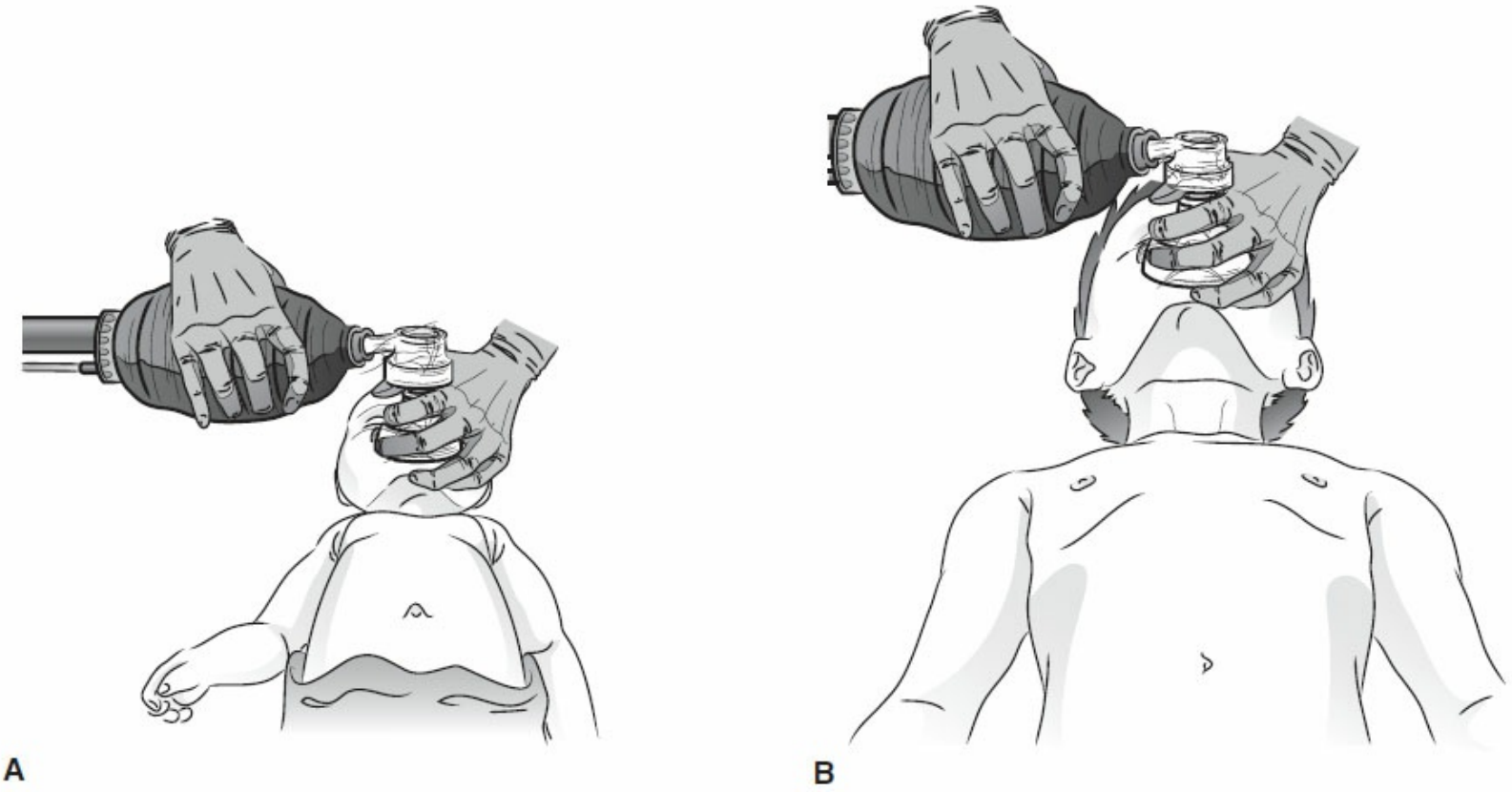
The 3 keys to providing adequate ventilation are:

- *Maintain an open airway.*
- *Establish a seal between the patient's face and the mask.*
- *Deliver optimal minute ventilation from the resuscitation bag to distal lung units.*

Positioning the patient appropriately is an essential first step. Maintaining a jaw thrust by positioning the last 3 fingers of 1 hand under the bony part of the mandible will facilitate ventilation. Keep these fingers off the soft tissue below the mandible to avoid occluding the airway. It may be necessary to move the patient's head and neck gently through a variety of positions to determine the optimum position for airway patency and effective ventilation. A neutral sniffing position without hyperextension is appropriate for infants and toddlers (ie, bring the face to the mask and not vice versa).

The technique for bag-mask ventilation is illustrated in **Figure 2-7**. The hand position is called the *E-C clamp technique*. The thumb and forefinger form a C shape and exert downward pressure on the mask while the remaining fingers of the same hand, forming an E, lift the jaw and pull the face toward the mask. This should create a tight seal. Once the mask is correctly applied, the other hand is used to compress the bag until the chest rises. Ventilation should synchronize with the patient's respiratory effort if the patient is spontaneously breathing; this will prevent gagging. It is better to lift the patient's face into the mask than to push the mask onto the patient's face.

Figure 2-7. Bag-Mask Ventilation



Note the E-C clamp technique as described in the text. Gentle cricoid pressure (**Figure 2-11**) applied by another rescuer may reduce gastric inflation and decrease the risk of aspiration during ventilation. Such pressure should be used only in an unconscious victim. Excessive pressure on the cricoid cartilage should be avoided; only 1 finger is necessary in a young child or infant.

Ventilation provided by 2 rescuers (**Figure 2-8**) can be more effective when there is significant airway obstruction or poor lung compliance. In this technique, 1 rescuer uses both hands to open the airway by placing 3 fingers of each hand on both posterior rami of the patient's mandible and maintains a tight mask-to-face seal while the other rescuer compresses the ventilation bag. Again, the rescuer holding the face mask should concentrate on lifting the jaw and face into the mask rather than pushing the mask onto the face.

Figure 2-8. Two-Person Bag-Mask Ventilation

To maintain a tight seal, avoid pushing the mask onto the face but instead use both hands to lift the mandibular rami anteriorly into the mask.

!

To assess the adequacy of bag-mask ventilation:

- *Evaluate chest rise and presence of bilateral breath sounds.*
- *Evaluate clinical response, including improved heart rate and return of good color.*
- *Monitor oxygen saturation.*

!

The 2-rescuer technique may be essential in children with difficult airways (eg, epiglottitis or foreign body aspiration with complete obstruction). If a patient has any respiratory effort at all, rescuer ventilations should be timed with them. Allowing expiration to occur fully before providing another breath avoids hyperinflation with consequent hemodynamic and oxygenation problems. Use only the force and tidal volume necessary to make the chest rise. Excessive volumes may compromise cardiac output, distend the stomach, increase the risk of vomiting and aspiration, and add to the risk of barotrauma.

E. Ventilation Bags

There are 2 types of ventilation bags: self-inflating and flow-inflating. Ventilation bags used for resuscitation should be self-inflating and a suitable size for the child. Neonatal bags (250 mL) hold only enough volume to be used with neonates. Pediatric bags (450-500 mL) are adequate for infants and children.

Many self-inflating bags are equipped with pressure-limited pop-off valves set at 35 to 40 cm H_2O. In patients with significant pulmonary disease or anatomic obstruction of the airway, these valves may need to be inactivated to achieve adequate chest rise. Flow-inflating bags require additional expertise. Pressures >20 cm H_2O should be avoided because of the risk of inflating the stomach and substantially reducing functional residual capacity.

F. Troubleshooting

Lack of adequate chest rise:

- Reposition the head. Make sure the head and neck are not hyperextended and thus causing airway obstruction.
- Ensure that the mask is the appropriate size and applied snugly on the patient's face.
- Make sure that you are lifting the child's jaw to the mask, not pushing the mask onto the child's face.
- Suction the airway if excessive secretions are noted.
- Place an OP airway.
- Disable the pop-off feature of the ventilator bag.
- Use the Sellick maneuver to decrease abdominal distension.
- Assess the need for a nasogastric tube to decompress the stomach and protect against aspiration.
- Check for the presence of a foreign body.

Adequate chest rise but low oxygen saturation:

- Check if the bag is connected to an appropriate oxygen source.
- Assess the need for higher pressures and disabling the pop-off valve.
- Consider using a positive end-expiratory pressure valve. Patients with lung disease might require additional pressure for improved oxygenation.
- In children with partial airway obstruction, application of 5 to 10 cm H_2O of continuous positive airway pressure may maintain adequate airway patency.

VI. ENDOTRACHEAL INTUBATION

Tracheal intubation is required when less-invasive methods of airway management prove inadequate. Many indications signal the need for intubation, but the most common is hypoxemic and/or hypercapnic respiratory failure. This is typically due to lower airway or parenchymal disease, such as bronchiolitis or pneumonia, and may result in failure of oxygenation, ventilation, or both. Upper airway obstruction caused by foreign bodies, infectious processes, tracheomalacia, tracheal stenosis, or extrinsic compression all may require intubation. Intubation also may be needed to provide cardiovascular and neurologic support. Indications for tracheal intubation are summarized in **Table 2-4**. See **Appendix 6** for further information on the procedure.

Table 2-4	INDICATIONS FOR TRACHEAL INTUBATION

- Respiratory failure
- Pao_2 <60 mm Hg (8.0 kPa) with fraction of inspired O_2 >0.6 (excludes cyanotic heart disease)
- $Paco_2$ >55 mm Hg (7.3 kPa)
- Excessive work of breathing
- Upper airway obstruction
- Hemodynamic instability (shock)
- Loss or inadequacy of airway protective reflexes
- Neuromuscular weakness
- Severe metabolic acidosis
- Head trauma (intracranial hypertension)
- Need for deep sedation and anesthesia
- Airway protection
- Therapeutic purposes

A. Tracheal Tubes

Tracheal tubes are sterile polyvinyl chloride tubes with a standard 15-mm adapter for attachment to a bag-mask device or ventilator tubing. They typically have centimeter markings on the side to document the depth of insertion as well as a vocal cord mark at the distal end to help approximate placement, although these are not uniform and should not be relied upon. Tubes are available in cuffed and uncuffed varieties. Historically, uncuffed tubes were recommended for children younger than 8 years because the trachea is narrowest at the cricoid ring in that age group. Uncuffed tubes allow a snug fit but avoid tracheal wall trauma from excessive cuff inflation. Cuffed tubes are constructed with a low-pressure cuff designed to minimize trauma to the tracheal mucosa. These tubes are very helpful in patients with severe lung disease who may require high peak inspiratory pressure or positive end-expiratory pressure. A cuffed tracheal tube should be inflated only to the minimum amount necessary to eliminate any air leak around the tube. In many cases, there will be no air leak, even with the cuff deflated.

Whether a tracheal tube is cuffed or uncuffed, proper fit is essential. When inserted, the tube should fit snugly into the trachea but should pass smoothly, without the need for excessive force. There are several methods of determining proper tube size based upon a patient's age and size. A commonly

used formula to calculate uncuffed tube size based on age is:

$$\text{Tube size (mm inner diameter)} = [\text{Age (years)}]/4 + 4$$

Cuffed tubes should be downsized by 0.5 mm when using this formula. The depth of insertion can be estimated by using the formula:

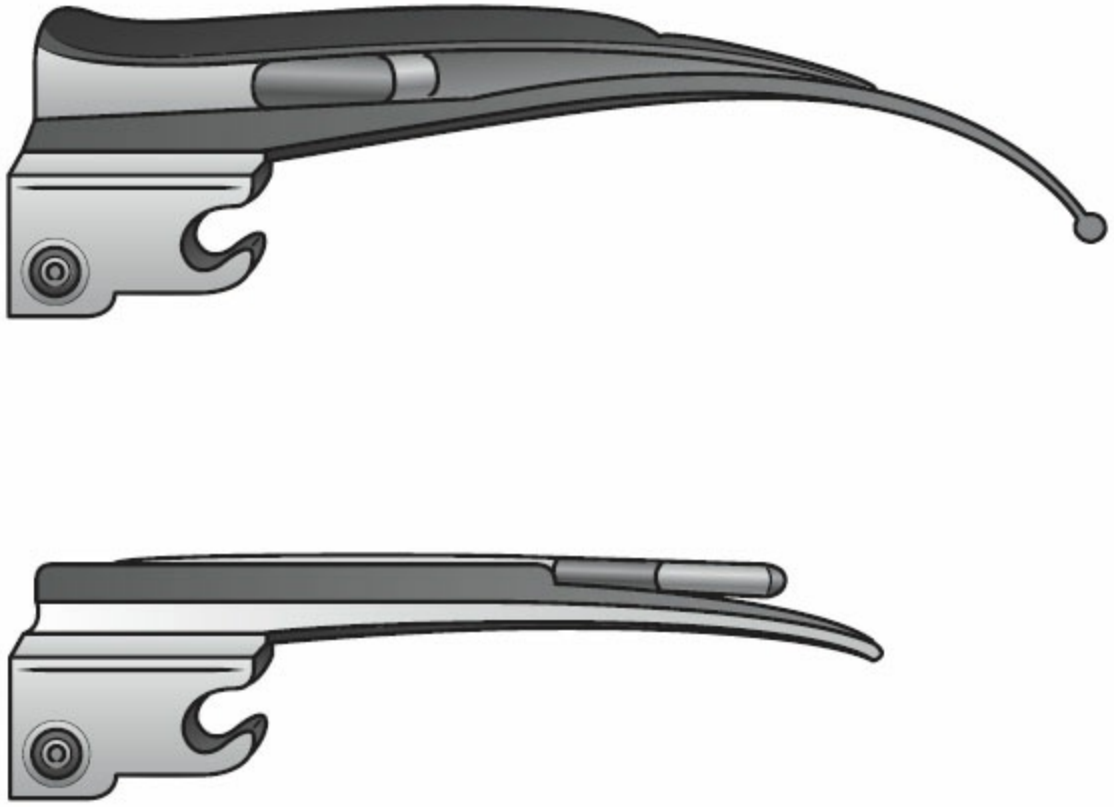
$$\text{Depth of insertion at lip} = \text{Inner diameter of selected tube} \times 3$$

These formulas yield estimates, and adjustments may be needed for individual patients. In all cases, tubes a size above and a size below the estimate should be available during intubation. Depth of insertion should be assessed by auscultation of breath sounds and a chest radiograph as soon as possible after intubation.

B. Laryngoscopes

The laryngoscope displaces the tongue and permits direct visualization of the larynx, vocal cords, and trachea. It consists of a handle (with battery) and a blade that has a light source at its tip. The 2 most common types of blades, the straight (eg, Miller, Robertshaw) and curved (eg, Macintosh) blades are shown in **Figure 2-9**. Each is used in a slightly different manner during intubation. Straight blades are typically used in infants and toddlers and are designed to directly lift the epiglottis out of the way, thus revealing the larynx and vocal cords. Curved blades are used in older children and adults, and are placed into the vallecula. Lifting the blade in the anterior direction indirectly lifts the epiglottis and reveals the vocal cords and related structures.

Figure 2-9. Laryngoscope Blades



The video laryngoscope incorporates a video camera into the device. A sensor at the tip of the laryngoscope blade transmits an image to a video monitor. The camera chip provides a high-quality image of the laryngeal structures. The image can be recorded and stored. The video laryngoscope can be used for both training and clinical purposes, especially in cases of a difficult airway.

C. Sedation and Analgesia

Most patients require sedation before laryngoscopy and intubation. The goal is to depress the level of consciousness sufficiently to produce appropriate conditions for intubation. Those conditions include adequate sedation, analgesia, and amnesia plus blunting of the physiologic responses to airway manipulation, all with a minimum of hemodynamic compromise. Factors that play a role in the selection of sedating agents include, but are not limited to, the agent's rapidity of onset, the patient's hemodynamic status, the need to prevent increases in intraocular or intracranial pressure that may be caused by intubation, and whether the patient is in a fasting state. In many cases, it is ideal to allow for the maintenance of spontaneous ventilatory drive during intubation. A wide variety of medications may be used for sedation, each with its own risks and benefits. In general, medications that act rapidly and are cleared quickly are best. Rapid clearance helps to limit the duration of potential complications. However, the short duration of action of some agents can result in a patient being intubated (which is painful and frightening) with inadequate sedation and analgesia. Thus, the child's level of consciousness should be continually assessed during and after intubation, with additional medication administered as appropriate. Providers should be familiar with the medications used at their institution so as to anticipate any side effects and be prepared to address them. A brief overview of some of the medications commonly used in tracheal intubation is presented in **Table 2-5**. (See **Appendix 7** for a more extensive review.)

Table 2-5 MEDICATIONS USED TO FACILITATE TRACHEAL INTUBATION

Agent	Dosing	Onset and Duration	Benefits	Cautions
Fentanyl	1-2 µg/kg IV bolus every 2 min titrated to effect	Onset: Immediate Duration: 30-60 min	• Rapid onset • Short acting • Reversible • Relatively stable hemodynamic profile	• Rigid chest syndrome • Respiratory depression • Lacks amnestic properties
Midazolam	0.05-0.1 mg/kg IV bolus every 5 min titrated to effect	Onset: 1-5 min Duration: 20-30 min	• Rapid onset • Short acting • Provides amnesia • Reversible	• Lacks analgesic properties • Respiratory depression • Hypotension and bradycardia
Ketamine	1 mg/kg IV bolus every 5 min titrated to effect	Onset: 1-2 min Duration: 10-30 min	• Rapid onset • Airway-protective reflexes remain intact • No hypotension or bradycardia	• Increases airway secretions and laryngospasm (blunted with atropine) • Elevates ocular pressure • Emergence reactions (blunted with benzodiazepines)

Etomidate	0.3 mg/kg IV bolus initially; 0.1 mg/kg bolus every 5 min thereafter, titrated to effect	Onset: 10-20 s Duration: 4-10 min	• Rapid onset • Short acting • Stable hemodynamic profile	• Potential for adrenal inhibition • May cause myoclonus • Not recommended for children under 10 y
Thiopental	2-3 mg/kg IV bolus, repeat as needed	Onset: 30-60 s Duration: 5-30 min	• Ultra short-acting barbiturate • Decreases intracranial pressure	• Cardiovascular and respiratory depression • Not approved by FDA for children
Propofol	1-3 mg/kg IV bolus initially; 0.5-2 mg/kg bolus every 3-5 min thereafter, titrated to effect	Onset: 30-60 s Duration: 5-10 min	• IV general anesthetic • Rapid onset and recovery	• Cardiovascular and respiratory depression • Contraindicated in patients with egg allergy

FDA, U.S. Food and Drug Administration

D. Neuromuscular Blockade

Patients who are inadequately relaxed despite adequate sedation may require neuromuscular blockade (or paralysis) as an adjunct to intubation. Therefore, it is vitally important to understand the power and risks of pharmacologic paralysis. Once neuromuscular blockers have been administered, all spontaneous respiratory effort ceases. Moreover, in patients with partial airway obstruction, neuromuscular blockade may worsen pharyngeal collapse, potentially resulting in complete airway obstruction. Airway patency must be maintained with bag-mask ventilation before neuromuscular blockade. If adequate chest rise and oxygen saturations cannot be readily sustained with bag-mask ventilation, neuromuscular blockers should not be given until a clinician highly skilled in advanced airway management is present. For the same reason, it is advisable to use, whenever possible, a rapidly acting and quickly cleared neuromuscular blocker. Some frequently used neuromuscular blockers are listed in **Table 2-6**.

Table 2-6 NEUROMUSCULAR BLOCKADE AGENTS

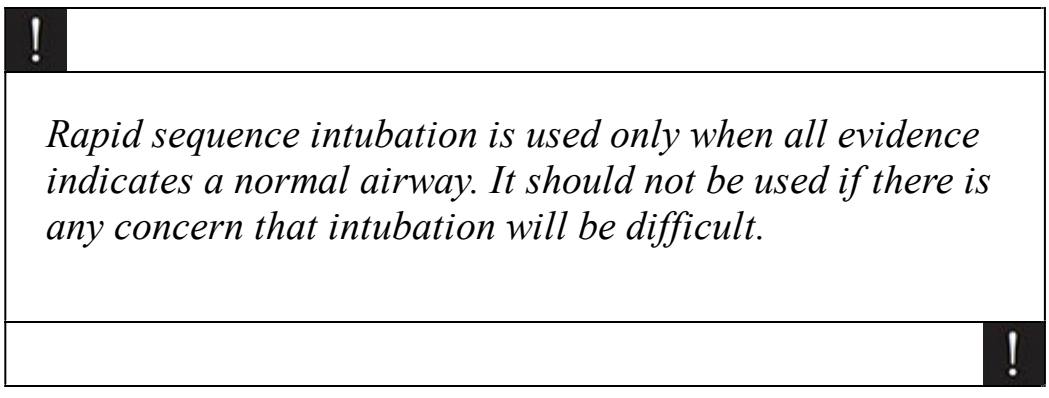
Agent	Dosing	Onset and Duration	Benefits	Cautions
Succinylcholine (depolarizing neuromuscular blockade)	1 mg/kg IV	Onset: 30-60 s Duration: 4-6 min	• Rapid onset • Short duration	• Causes muscle fasciculation (blunted with low-dose nondepolarizing neuromuscular blocker) • Potentiates hyperkalemia (contraindicated in head trauma, crush injury, burns, hyperkalemia) • May induce neuroleptic

				malignant syndrome
Vecuronium (nondepolarizing neuromuscular blockade)	0.1 mg/kg IV	Onset: 1-3 min Duration: 30-40 min	• No fasciculations	• Slow onset • Longer duration of action
Rocuronium (nondepolarizing neuromuscular blockade)	0.6-1.0 mg/kg IV	Onset: 30-60 s Duration: 30-40 min	• No fasciculations	
Cisatracurium (nondepolarizing neuromuscular blockade)	0.1-0.2 mg/kg IV	Onset: 2-3 min Duration: 35-45 min	• Can be used in renal failure • Metabolized through Hoffman elimination	• Potential histamine release
Atracurium (nondepolarizing neuromuscular blockade)	0.3-0.5 mg/kg	Onset: 3-5 min Duration: 20-35 min	• Can be used in renal failure • Metabolized through Hoffman elimination	• Potential histamine release

As with sedatives, it is necessary to be familiar with the pharmacology and side effects of neuromuscular blocking agents prior to using them. Succinylcholine (suxamethonium chloride), in particular, can cause hyperkalemia and malignant hyperthermia. Neuromuscular blockers provide no sedation or analgesia. Long-acting blockers, like vecuronium, often have a more extensive duration of action than the sedative and analgesic medications given before intubation. Additional sedation should be provided at regular intervals to ensure that the patient does not awaken during neuromuscular blockade.

E. Rapid Sequence Intubation

Rapid sequence intubation is employed when there is concern about aspiration (eg, full stomach) and no suspicion of a difficult intubation. The goal is to obtain airway control with a tracheal tube as quickly as possible, thus minimizing aspiration risk. Preoxygenation with a nonrebreathing mask increases the available oxygen in the lungs during the procedure. When all necessary intubation equipment and personnel are prepared, rapidly acting sedative, analgesic, and paralytic drugs are given simultaneously. Cricoid pressure is applied at the outset and maintained until the trachea is intubated and adequate confirmatory maneuvers are completed.


!
Rapid sequence intubation is used only when all evidence indicates a normal airway. It should not be used if there is any concern that intubation will be difficult.
!

F. Other Intubation Equipment

Appropriate intubation supplies and equipment (**Table 2-7**) should be assembled at the bedside before beginning an intubation procedure. Cardiopulmonary monitoring and pulse oximetry should be used throughout the procedure. Suction equipment with a large-bore, rigid device (such as a Yankauer suction tip) should be used to remove mucus, blood, and particulate matter from the oropharynx. A flexible suction catheter to use on the tracheal tube is helpful as well.

Table 2-7 INTUBATION SUPPLIES AND EQUIPMENT

Supplies	Comments
Cardiopulmonary monitor and pulse oximeter	Monitor continuously before, during, and after intubation
Suction devices	Test oral (Yankauer) and tracheal tube suction beforehand
Stylet	Ensure appropriate size and ease of removal
Bag, face mask, and oxygen supply	Test size and function beforehand
Oro- or nasopharyngeal airway	
Tracheal tubes	Have size above and size below the estimated size at bedside
Syringe, 3 mL, to inflate the endotracheal tube cuff	Ensure cuff is functioning properly
Laryngoscope	Ensure light is functioning properly; have various sizes and types of blades at bedside (Miller and Macintosh)
End-tidal CO_2 detector	Turns yellow when CO_2 detected
Magill forceps	Use for grasping and advancing tracheal tube during nasotracheal intubation
Oxygenating stylet	May be used to insufflate oxygen and as guide for tracheal tube placement
Tape or tube-securing device	Adhesive
Stethoscope	
Medications for intubation, if indicated	
Capnography (not colorimetric capnometry)	Universal standard of care outside combat zones

A stylet may be used to add rigidity to the tracheal tube. To avoid airway trauma, ensure that the stylet does not protrude from the distal end of the tube. Also, determine that the stylet can be removed easily once it is in position. Many successfully placed tracheal tubes have become dislodged in the process of removing tight stylets.

An end-tidal CO_2 detector should be attached to the tracheal tube after intubation to confirm proper placement. Colorimetric end-tidal CO_2 devices change color from purple to yellow to confirm the presence of exhaled CO_2 and tracheal placement. Accuracy can be affected by gastric acid and bicarbonate. The endotracheal tube should be secured with tape or a tracheal tube-securing device with attention to the depth; inadvertent displacement can compromise oxygenation and ventilation.

G. Intubation

All age-appropriate supplies and equipment should be assembled and checked for proper functioning before attempting an intubation. Various sizes and shapes of laryngoscope blades should be readily available. Once endotracheal tube size is selected, additional tubes that are one-half to one full size smaller and larger should be available. Proper positioning is a key element of successful intubation. To visualize the glottis clearly, the oral, pharyngeal, and tracheal axes must be brought into alignment as described previously and shown in **Figure 2-3**. Alignment of the 3 airway axes can be achieved with slight hyperextension of the head into the sniffing position, with the ear canal just above or even with the top of the shoulder when visualized from the side.

Children younger than 2 years have relatively large heads for their body size, which results in slight neck flexion when they are in the supine position. These children usually benefit from a small roll (thin towel or blanket) placed under the shoulders, which allows the head to fall back into the proper position but avoids hyperextension. In older children, a small roll under the occiput helps facilitate proper alignment of the axes. In patients with suspected cervical spine injury, the head should remain in the neutral position at all times and the spine should be stabilized while intubating.

When possible, it is advantageous to preoxygenate patients to 100% oxygen saturation with bag-mask ventilation before attempting intubation. Each laryngoscopy and tracheal tube placement attempt should be limited to approximately 30 seconds. An assistant should monitor vital signs continuously. All attempts must be expedient, and failed attempts should be terminated before desaturation. After each unsuccessful attempt, the patient should again receive bag-mask ventilation to optimize oxygenation before the next attempt. Multiple attempts at intubation may lead to airway and vocal cord edema, which may further compromise ventilation and intubation.

Not all intubation attempts will be successful, and anticipation of a difficult airway is a vital part of the assessment before intubation is attempted. A history of difficult intubation, obstructive sleep apnea, or frequent snoring should raise concern about the level of difficulty to be expected. Patients with facial abnormalities such as micrognathia, midface hypoplasia, small mouths, large tongues, and morbid obesity often are difficult to intubate. Limited mobility of the temporomandibular joint or the cervical spine, as well as bleeding, masses, or foreign bodies in the upper airway, all present challenges to tracheal intubation.

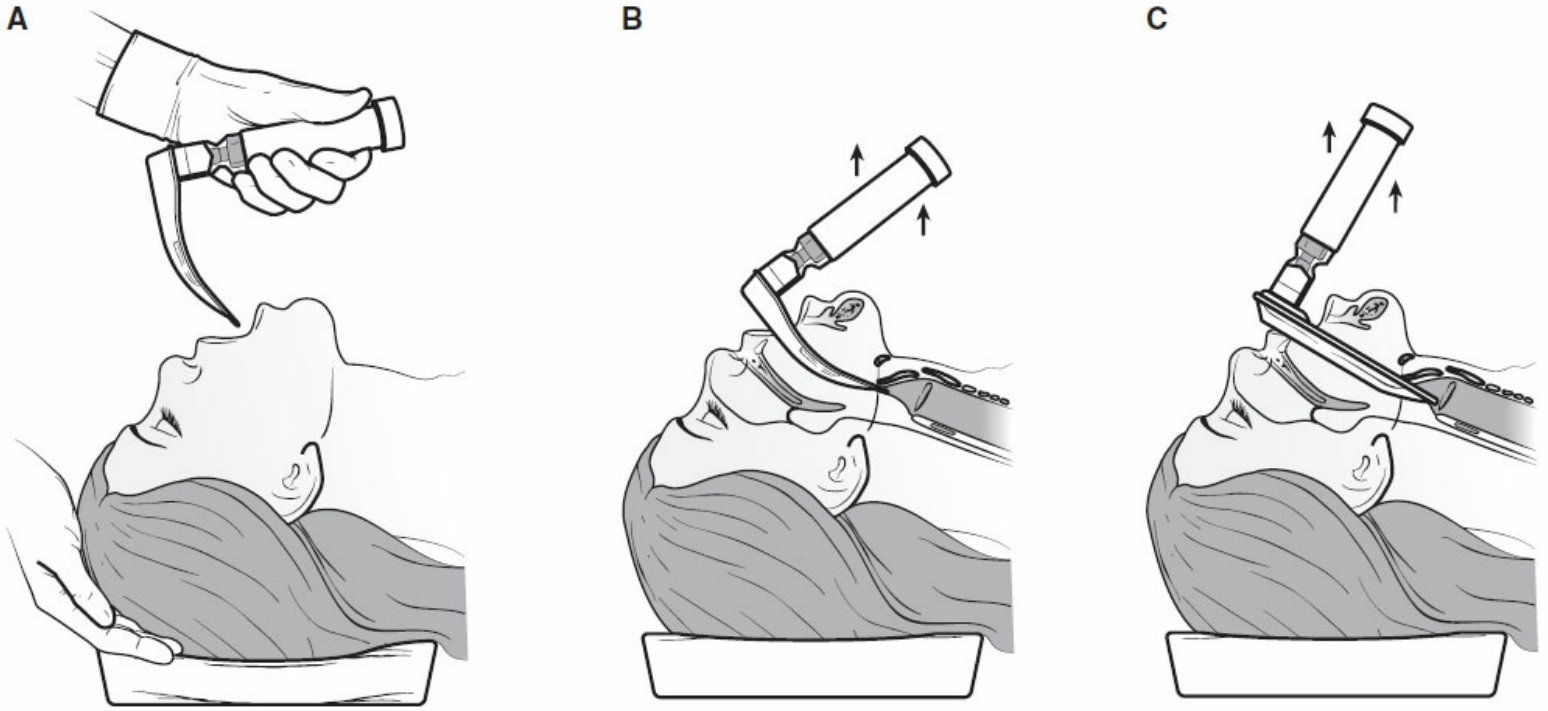
A backup plan should always be developed prior to intubation. Supplies should be available for multiple attempts, advanced intubation techniques, and cricothyrotomy (**Table 2-7**). A strategy for management of the “cannot ventilate, cannot intubate” scenario should be developed (**Appendix 8**). If a difficult intubation is anticipated and patient stability permits, it is advisable to have experienced clinicians present before proceeding. Consultation with a pediatric anesthesiologist or otolaryngologist should be considered.

Orotracheal intubation is the most common and preferred method of intubation in emergency situations. The technique is summarized in **Table 2-8** and shown in **Figure 2-10**.

Table 2-8	TECHNIQUE FOR OROTRACHEAL INTUBATION

- Ensure that the patient is positioned correctly.
- Hold the laryngoscope in the left hand and advance the blade along the right side of the mouth to the base of the tongue.
- Sweep the blade to the midline, thus lifting the tongue out of the visual field and providing an area on the right where the tube can be advanced without losing direct visualization of the laryngeal structures. Ensure that the tongue is properly displaced to ensure optimal visualization and facilitate successful intubation.
- When using a straight blade, position the distal end of the blade just below (posterior to) the epiglottis so it may be used to lift the epiglottis anteriorly. Often, a straight blade will enter the esophagus upon insertion. If the blade tip is in the esophagus, slowly backing out the blade will cause the larynx to fall into view as the blade leaves the esophageal inlet. When a curved blade is used, the end of the blade is placed into the vallecula above (anterior to) the epiglottis. Straight blade and curved blade insertions are illustrated in **Figure 2-10**.

Figure 2-10. Insertion of Laryngoscope Blades



A. Head position prior to laryngoscope insertion with a curved blade
B. Position of the laryngospcope in the airway with a curved blade
C. Position of the laryngoscope with a straight blade

Insertion techniques are summarized in **Table 2-8**.

The anterior placement of the pediatric airway sometimes makes visualization of the epiglottis and larynx difficult. Slight pressure on the cricoid cartilage (Sellick maneuver) may move the larynx and the airway posterior, and improve visualization (**Figure 2-11**). The Sellick maneuver should not be confused with laryngeal manipulation. The Sellick maneuver invariably worsens the view; backward, upward, and rightward pressure (mnemonic *BURP*) improves it.

Figure 2-11. Cricoid Pressure (Sellick Maneuver)

Gentle pressure on the cricoid cartilage by a second person may reduce gastric inflation and decrease the risk of aspiration during bagging. In an infant or child, cricoid pressure can also improve an intubator's view of the larynx.

Once either a straight or curved blade is properly positioned, the laryngoscope handle should be lifted anteriorly along an axis that is approximately 45° from the bed, providing a clear view of the larynx. Do not lever the blade upwards, as this will result in trauma to the lips, gums, and teeth from the proximal end of the blade and the handle. Remember, even in an edentulous infant, teeth are developing right below the gum line.

Once the larynx is visualized (**Figure 2-12**), maintain visualization while an assistant passes the necessary equipment. Advance the tube from the right corner of the mouth through the vocal cords. Do not advance the tube down the flange of the blade because doing so will obscure the larynx. A good rule of thumb is that if the operator does not see the tube go through the vocal cords, it has not done so.

Figure 2-12. Visualization of the Larynx during Laryngoscopy

Courtesy of Sanjay R. Parikh, MD FACS, Seattle Children's Hospital, University of Washington.

The tip of the tracheal tube should be placed at the mid-tracheal level. Cuffed tubes should be positioned with the cuff just below the vocal cords.

Once the tube has been inserted, confirm proper tracheal placement with an end-tidal CO_2 detector. Look for a symmetric chest rise and listen for equal breath sounds while delivering positive pressure breaths. If the tube is in the right main-stem bronchus, breath sounds will be more prominent over the right chest, signaling that the tube needs to be repositioned. Slowly pull the tube back while listening to the left chest. When breath sounds become audible on the left, the tube has entered the trachea. Listen over the stomach; if the tube is in the trachea, breath sounds will be absent over the abdomen. Breath sounds are easily transmitted across the chest wall in smaller children, so smaller tidal volumes should be used during auscultation for verification of tube placement. Condensation in the tracheal tube suggests, but does not confirm, tracheal placement.

During cardiac arrest or when cardiac output is very low, minimal blood flows to the pulmonary capillaries and, consequently, very little to no CO_2 is delivered to the lungs for exhalation. In such cases, CO_2 may not be detected despite proper tube placement, so other methods of confirmation are necessary. Because no single method is completely dependable in all situations, multiple methods of confirmation should always be used. If any question exists, perform laryngoscopy to obtain visual confirmation. A chest radiograph should be acquired as soon as possible to ensure proper position and to evaluate for complications such as pneumothorax.

The tracheal tube should be secured to the patient's face to avoid unintentional extubation. This can be achieved with adhesive (eg, tincture of benzoin) and two pieces of tape partially ripped so that each resembles a pair of pants. The "waist" of the tape is placed on the patient's cheek, one leg is placed above the patient's upper lip, and the other is tightly wrapped around the tube. The other piece of tape is placed below the lower lip in the same manner. Commercially manufactured tube holders are also available.

Following intubation, monitoring by electrocardiography, pulse oximetry, and end-tidal CO_2 should be continued. Once a patient is connected to a ventilator, an arterial blood gas study should be obtained as soon as possible.

H. Possible Complications of Endotracheal Intubation

The physiologic alterations that occur when positive pressure ventilation is initiated can lead to complications; however, these can be anticipated and rapidly treated. As noted previously, bag-mask ventilation delivers some of the volume from each positive pressure breath to the stomach, leading to distention (which may make it difficult to adequately ventilate the patient) and aspiration. To decrease the likelihood of aspiration, a naso- or orogastric tube may be placed to help decompress the stomach and remove stomach contents.

Stimulation of the airway results in activation of the vagus nerve. When a patient is intubated, this appears clinically as bradycardia. Atropine (0.01-0.02 mg/kg; maximum single dose, 1 mg; minimum single dose, 0.1 mg) may be administered to overcome this response. In patients who are bradycardic at baseline, or in whom there is concern for vagal-mediated bradycardia, atropine may be given before attempting intubation, particularly if a straight blade is being used and the dorsal (vagally innervated) side of the epiglottis is being manipulated. Atropine is also used to decrease airway secretions in patients, especially those who receive ketamine for sedation.

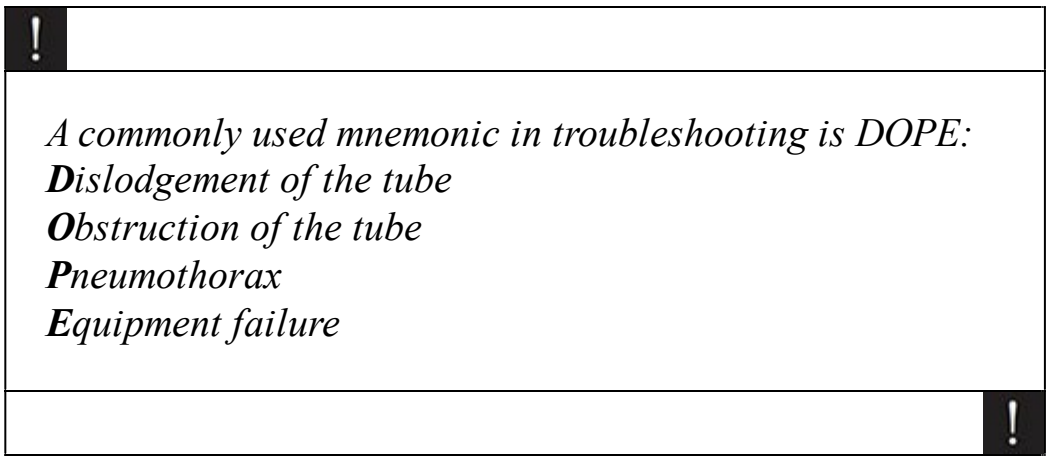
Positive pressure ventilation has the potential to cause barotrauma to the alveoli, occasionally resulting in leakage of air into the pleural space (pneumothorax). As air accumulates, the lung is compressed, compromising oxygenation and ventilation. If the air leak is ongoing, the pressure in the pleural space rises, causing a tension pneumothorax. This shifts the mediastinum toward the uninvolved side, impairing venous return to the heart and compromising cardiac output. Signs of tension pneumothorax include deviation of the trachea away from the affected side, diminished breath sounds and hyperresonance to percussion on the affected side, tachycardia, and falling blood pressure. Treatment should not be delayed to obtain a chest radiograph. A tension pneumothorax can be decompressed by the sterile passage of an appropriately sized angiocatheter into the pleural space over the top of the third rib in the midclavicular line or by finger thoracostomy in larger children. A rush of air will be heard as the air under pressure escapes the pleural space. Definitive treatment is with a thoracostomy tube, which should be placed as soon as possible.

Another complication of positive pressure ventilation is decreased cardiac output. The increase in intrathoracic pressure impairs venous return to the right side of the heart, thus decreasing preload. In patients who are dehydrated and have compromised hemodynamic function, this impairment may produce clinically relevant decreases in cardiac output and hypotension. A reduction in tidal volume, along with fluid resuscitation, will often restore preload and improve cardiac output.

I. Troubleshooting After Intubation

Acute respiratory deterioration can occur in an intubated patient for a variety of reasons. A commonly used mnemonic in this situation is *DOPE*: dislodgement of the tube, obstruction of the tube, pneumothorax, and equipment failure. View chest rise and auscultate the chest to ensure proper

positioning of the tracheal tube. Asymmetric breath sounds may represent improper tube position or pneumothorax. When asymmetry is present, a chest radiograph can help identify the problem. Suction the tube to clear secretions from the lumen. If the patient is on a ventilator, check that it is functioning properly. In cases of uncertainty, it may be helpful to remove the patient from the ventilator and ventilate manually with 100% oxygen.



Respiratory deterioration may be secondary to the patient's underlying condition. Adjustments to the ventilator and other respiratory therapies may be required to improve the child's respiratory status.

VII. DIFFICULT AIRWAY

Sometimes intubation is unachievable due to complete upper airway obstruction, anatomic airway abnormalities, severe facial trauma, or similar circumstances. It is important to have a backup plan in the event that a tracheal tube cannot be placed. First and foremost, bag-mask ventilation may be provided until a definitive airway is established. See **Appendix 5** for additional information on airway adjuncts.

A. Laryngeal Mask Airway

When it is impossible to intubate or manually ventilate a patient by mask, a laryngeal mask airway may provide an open airway and permit gas exchange. A laryngeal mask airway consists of a tube with a cuffed, mask-like projection at the distal end. When properly placed, it sits just above the glottic opening. These airways come in sizes appropriate for children. A laryngeal mask airway does not protect against aspiration and should not be used as a definitive airway.

The weight range for the size of a given laryngeal mask airway is printed on the mask itself. **Appendix 5** shows the insertion technique. The airway is held like a pencil with the distal opening facing away from the operator (so that it ends up facing anteriorly when placed). The balloon is placed against the hard palate, which is used as a guide as the airway is inserted. Once definite resistance is felt, the balloon is inflated and bag ventilation can be initiated. Assessment of air entry is the same as for intubated patients, with attention to symmetric lung inflation. Ideally ventilation uses only enough force to move the chest wall adequately, which may be assessed by auscultating the chest for normal respiratory sounds. Higher ventilation pressures may lead to insufflation of the

stomach in addition to the lungs.

A special type of laryngeal mask airway, the intubating laryngeal mask airway, is commercially available for use in larger patients. It has a bar at the distal end that elevates the epiglottis and is wide enough to accommodate a special tracheal tube passed through the lumen. Once in position, a tracheal tube is passed through the laryngeal mask airway. The bar is forced up, lifting the epiglottis and allowing the tracheal tube to be passed into the trachea. The intubating laryngeal mask airway may then be removed over the tracheal tube (although this is not necessary), which is left in position.

B. Esophageal-Tracheal Double-Lumen Airway Device

In children taller than 4 feet, an esophageal-tracheal double-lumen airway device can be placed. These tubes contain two lumens as well as proximal and distal balloons. They are placed blindly and almost always end up in the esophagus. Both balloons are then inflated to create a seal. (See **Appendix 5** for insertion technique.)

When properly positioned, the distal balloon obstructs the esophagus and the proximal balloon obstructs the distal pharynx. Holes in the proximal tube are above the distal balloon. Thus, air is forced out of the proximal tube down the trachea. If the tube ends up in the trachea, the distal tube may be used to ventilate the patient. A similar device, a laryngeal tube with a single lumen, is available in multiple sizes.

C. Intubating Stylets

When a patient's vocal cords are visualized but the tracheal tube cannot be passed, it may be easier to pass an intubating stylet. These stylets are hollow and have a connector at the end that can be attached to an oxygen source or a bag-mask device. Once in place, they can be used to ventilate the patient by hand. A tracheal tube can then be passed over the stylet, and the stylet can be removed, leaving the tracheal tube in place.

D. Bougie

This can be used as an introducer for difficult airways. The standard size is 15F, 60 cm. The pediatric size is 10F, which can be used for airways with diameters between 4 and 6 mm. It can be inserted after direct visualization of the larynx. In the situation where the larynx is not visualized, it can be inserted into the trachea blindly. This can then be used as a guide for insertion of the endotracheal tube.

E. Fiberoptic Intubation

Operators familiar with the use of intubating bronchoscopes can provide assistance with a difficult airway. The endotracheal tube is loaded onto the bronchoscope, and the bronchoscope is placed into the trachea through the vocal cords under direct visualization. The endotracheal tube is then slipped

off the end of the bronchoscope and left in proper position within the trachea.

F. Cricothyrotomy

In extreme situations, cricothyrotomy may be performed. With the patient's neck hyperextended, the cricothyroid membrane is located by palpation between the thyroid cartilage and the cricoid cartilage. The area is sterilized, and an IV catheter (catheter over needle) is placed through the cartilage into the trachea. Proper completion of this step is confirmed by the patient's aspiration of air, after which the needle is withdrawn while advancing the catheter. The catheter can then be attached to a positive pressure device via the Luer-Lok connection. A 5-mL syringe barrel (with the plunger removed) will accept a standard 15-mm adapter, and manual ventilation can be performed. Commercially available cricothyrotomy kits contain all the necessary supplies. Although these kits are potentially useful in older children, the unique anatomy of the infant's larynx, the small size of the cricothyroid membrane, and the technical difficulty in locating the correct anatomical structures make their use impractical, if not dangerous, in neonates and infants. Consultation with a pediatric otolaryngologist should be considered in situations where a cricothyrotomy or emergency tracheostomy is necessary.

G. Planning for a Difficult or Failed Intubation

Whenever an airway proves difficult to intubate, or when difficulty is anticipated, backup personnel with expertise in the management of the pediatric airway should be summoned. Anesthesiologists, otolaryngologists, and pediatric intensivists are considered experts by training in difficult airways. An algorithm for managing a difficult intubation is presented in **Appendix 8**.

Key Points

Airway Management

- Accurate assessments of the work of breathing, airway patency, respiratory drive, and consciousness are crucial first steps in the management of children with breathing difficulty.
- Maintaining a patent airway via manual methods (eg, oropharyngeal and nasopharyngeal airways) is an important skill in airway management.
- All healthcare providers for critically ill children should be able to supply adequate oxygenation and ventilation with bag-mask ventilation.
- All necessary supplies and personnel must be present before intubation is attempted.
- Expertise with sedatives, analgesics, and neuromuscular blockers is a prerequisite for intubation.

- A backup plan should be developed before a potentially difficult intubation is attempted. The use of airway adjuncts, such as a laryngeal mask airway, and cricothyrotomy may be lifesaving when intubation cannot be achieved.

Suggested Readings

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PEDIATRIC CARDIAC ARREST

Objectives

- Rapidly identify the need for cardiopulmonary resuscitation (CPR) to prevent or treat cardiac arrest.
- Understand the key components of high-quality CPR.
- Understand the physiology of CPR.
- Assess and initiate high-quality, goal-directed post-resuscitation care for infants and children following cardiac arrest.

Case Study

A 5-year-old boy with no prior medical history presents to an emergency department in severe respiratory distress following an accidental drowning in a swimming pool. The pulse oximeter saturation is 76% on room air. During your examination, the child becomes apneic, bradycardic, and unresponsive to vigorous stimulation. Bag-mask ventilation and chest compressions are initiated.

Detection

– What are the key findings in the initial assessment?

– What is the likely diagnosis?

Intervention

– With CPR ongoing, what is the most important next step?

Reassessment

– When is the appropriate time to reassess the patient's respiratory effort and circulation?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to care for this patient?

Teamwork

– How will the treatment strategy be implemented?

– What is the composition of the medical team who can best manage this child at this stage?

– Who is to do what and when?

I. INTRODUCTION

Successful resuscitation after pediatric cardiac arrest depends upon rapid identification and treatment of the cause of the arrest, immediate initiation of high-quality cardiopulmonary resuscitation (CPR), and aggressive post-resuscitation care. Critical factors that influence survival outcomes include the environment in which the arrest occurs, the child's preexisting condition, the duration of no pulsatile flow prior to resuscitation, the initial electrocardiographic (ECG) rhythm detected, the quality of the basic and advanced life support interventions provided during the arrest, and the meticulous attention to selected post-resuscitation support parameters (eg, temperature, oxygen, ventilation, blood pressure, glucose, seizure control).

Pediatric in-hospital CPR survival rates are relatively good. Approximately three-quarters of in-hospital pediatric arrest patients achieve return of spontaneous circulation (ROSC), more than one-third of whom survive to hospital discharge. Most survivors (>75%) have a favorable neurologic outcome.

Outcomes following pediatric out-of-hospital arrests are not as good; survival to hospital discharge typically occurs in less than 10% of these children, and many have severe neurologic sequelae. These poor outcomes are due in part to prolonged periods of no pulsatile flow because many of these cardiac arrests are not witnessed, and only 30% to 50% of children receive bystander CPR. In the first year of life, sudden infant death syndrome is the leading cause of out-of-hospital pediatric cardiac arrest, followed by trauma, airway obstruction, and drowning.

II. PREVENTION/RECOGNITION

The most common causes of in-hospital cardiac arrest are acute respiratory insufficiency or circulatory shock. Patients often have abnormal physiologic parameters in the hours before the event. Mortality and morbidity rates can be decreased in the prearrest phase with the utilization of the DIRECT algorithm (**Chapter 1**).

Reassessment and review of trends in vital signs and response to therapy are critical. Recognition of end-organ perfusion abnormalities, such as oliguria, altered mental status, and delayed capillary refill, may be hallmarks of impending failure. Gasping, bradycardia, and severe lethargy (eg, loss of consciousness) are late signs of shock and impending cardiac arrest.

Early warning scores (**Chapter 1**) and the rapid response of medical emergency teams are in-hospital emergency systems designed to identify and respond to patients in imminent danger of decompensation, thereby preventing progression to cardiac arrest. Implementation of a pediatric rapid response team has been associated with a lower rate of cardiac arrest outside of the pediatric ICU. Rapid response teams cannot prevent all cardiac arrests, but they are able to transfer critically ill children to an ICU for better monitoring and more aggressive interventions to prevent arrest or to provide prompt advanced life support if the arrest occurs. The implication of this paradigm is that any cardiac arrest that occurs outside a monitored unit should be reviewed as a potentially avoidable serious safety event.

III. DIAGNOSIS OF CARDIAC ARREST

Lack of movement or response to vigorous stimulation, without signs of effective breathing (eg, no normal breathing other than an occasional gasp), should be treated as a cardiac arrest until proven otherwise. Palpation of a central pulse may not be reliable in emergency situations. If more advanced monitoring is in place at the time of arrest (eg, pulse oximetry, exhaled carbon dioxide, arterial catheter), this can be used to support the diagnosis.

The cause of the arrest should be determined in the early minutes of resuscitation. A focused physical exam should be conducted and a brief medical history obtained. A cardiopulmonary monitor should be placed and the ECG should be examined. A blood gas analysis with electrolytes (Na^+, K^+, Ca^{2+}) and bedside glucose level should be performed with point-of-care testing, if available.

A. Pulse Check

The triad of pulselessness, apnea, and unresponsiveness defines the clinical state of cardiac arrest. Healthcare providers should spend no longer than 10 seconds assessing a central pulse. The empiric provision of chest compressions without a pulse check for the child who “appears lifeless” (unresponsive to vigorous stimulation, not breathing normally) is appropriate.

B. Breathing

For decades, treatment recommendations by the International Liaison Committee on Resuscitation and American Heart Association (AHA) guidelines used the mnemonic ABC (airway, breathing, circulation) for the stepwise assessment and intervention sequence in suspected cardiac arrest. With the 2010 change from ABC to CAB (compressions, airway, breathing), the first steps to resuscitation no longer involve spending up to 10 seconds assessing breathing. Instead, the patient’s general appearance, with attention to a lack of effective breathing, must be assessed immediately to determine whether chest compressions are indicated. Irregular or agonal respirations (eg, gasping) should not be interpreted as an effective respiratory pattern; an unresponsive patient with agonal respirations requires immediate chest compressions and initiation of effective CPR.

IV. MECHANISM OF BLOOD FLOW

A. Physiology of Coronary Blood Flow

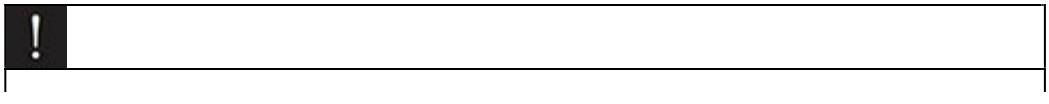
The coronary arteries provide blood flow from the aortic root to the myocardium. The normally beating heart is mainly perfused by coronary blood flow during diastole (heart muscle relaxation). When the heart arrests and no blood flows to the aorta, coronary blood flow ceases. During chest compressions, aortic pressure rises at the same time as right atrial pressure. During the decompression phase, the right atrial pressure falls faster and lower than the aortic pressure, generating a pressure gradient that perfuses the heart with oxygenated blood. Coronary perfusion pressure below 15 mm Hg during CPR is a poor prognostic factor for ROSC, so it is important to employ effective chest compressions and minimize interruptions during CPR.

B. Physiology of Ventilation

The importance of negative intrathoracic pressure on venous return during CPR, and thereby on coronary perfusion pressure and myocardial blood flow, has been rediscovered. During the decompression phase (chest compression release), negative intrathoracic pressure allows venous return to the heart and improves coronary artery blood flow. This can be experimentally enhanced either by active decompression (suction upwards, manual or automated) or by briefly impeding air flow to the lungs (ie, with an inspiratory impedance threshold device). Increased negative pressure promotes venous return, cardiac output, and mean aortic pressure.

V. CARDIOPULMONARY RESUSCITATION

In 2010, the AHA made the recommendation to change the algorithmic sequence of rescue interventions for the arrested patient from ABC to CAB (compressions, airway, breathing). The change was made because blood flow during cardiac arrest depends on chest compressions, and efforts to address airway and breathing delay the reestablishment of blood flow. The value of this approach is most dramatically demonstrated by the success of compression-only CPR (ie, without rescue breathing) in adults. Although the CAB sequence is the adult recommendation, it is reasonable to use the same approach in children to simplify training for the lay rescuer, especially when the arrest is sudden and witnessed (ie, has a higher likelihood of a cardiac etiology). Additionally, starting pediatric resuscitation with compressions instead of ventilation results in only a brief delay in the first rescue breath. Furthermore, for in-hospital events, given that chest compressions can be applied instantaneously and positive-pressure ventilation requires equipment preparation, the CAB approach may actually enhance initiation of CPR. The European Resuscitation Council guidelines have retained with ABC approach, but the emphasis remains on minimizing any delay in initiating chest compressions.



The algorithmic sequence of rescue interventions for the arrested patient is CAB (compressions, airway, breathing).

The CAB sequence applies to the patient presumed to be in cardiac arrest. In cases of respiratory compromise leading to hypoxemia and bradycardia, attention to airway, breathing, and oxygenation before arrest can be lifesaving and is recommended.

A. Circulation

Basic life support with early, effective, and continuous chest compressions is generally the best way to provide circulation during cardiac arrest. The most critical elements are to push hard and push fast. Because there is no flow without chest compressions, it is important to minimize interruptions in compressions. To facilitate good venous return in the decompression phase, allow full chest recoil between compressions and avoid excessive ventilation. The latter can impede venous return because of increased intrathoracic pressure.

Keys to High-Quality Cardiopulmonary Resuscitation

- *Push hard*
- *Push fast (approximately 100-120 compressions per minute)*
- *Minimize interruptions*
- *Allow full chest recoil*
- *Avoid excessive ventilation (approximately 10 breaths per minute)*

Excellent closed-chest CPR generates approximately 10% to 25% of baseline myocardial blood flow and a cerebral blood flow that is approximately 50% of normal. By contrast, open-chest CPR can generate a cerebral blood flow that approaches normal. Although open-chest cardiac compression improves coronary perfusion pressure and increases the chance of successful defibrillation, surgical thoracotomy is impractical in many situations and increases cost without long-term survival benefit. Early consideration of open-chest CPR may warrant consideration in select resuscitation circumstances (eg, penetrating trauma, postcardiac surgery).

B. Airway

While securing the airway is important, tracheal intubation requires a care provider with advanced airway skills and often delays or interrupts the initiation of chest compressions. Because effective blood flow is critical to achieving ROSC, compressions or defibrillation must be initiated immediately, along with effective ventilation, most frequently with bag-mask technique. Tracheal intubation for ventricular fibrillation (VF) may not be necessary, and defibrillation should be prioritized to ensure the best chance of successful conversion to a perfusing rhythm. Effective ventilation should be high priority, minimally interrupting chest compressions to place an advanced airway.

1. Delayed Intubation with Ventricular Fibrillation

During sudden witnessed collapse (VF cardiac arrest), an oxygen reserve remains in the lungs. Acceptable Pao_2 and $Paco_2$ levels persist 4 to 8 minutes during chest compressions without rescue breathing. Aortic oxygen and carbon dioxide concentrations do not change from prearrest levels, even without chest compressions, because no blood flows and aortic oxygen consumption is minimal. Adequate oxygenation and ventilation can continue without rescue breathing because the lungs serve as a reservoir for oxygen during the low-flow state of CPR. In contrast, during asphyxia, which is the more common etiology of pediatric cardiac arrest, blood continues to flow to tissues; therefore, arterial and venous oxygen saturations decrease while carbon dioxide and lactate levels increase. In addition, continued pulmonary blood flow before the arrest depletes the oxygen reservoir. Therefore, rescue breathing is critical for cardiac arrest preceded by asphyxia because significant arterial hypoxemia and acidemia exist prior to initiation of CPR.

C. Breathing

1. Ventilation and Compression-Ventilation Ratios

Physiologic estimates suggest the amount of ventilation needed during CPR is much less than that needed during a normal perfusing rhythm because the cardiac output during CPR is only 10% to 25% of that during normal sinus rhythm. A chest compression-to-ventilation ratio of 15:2 delivers the same minute ventilation as CPR with a ratio of 5:1, but the number of compressions delivered is 50% higher with the 15:2 ratio (**Table 3-1**).

Table 3-1 RESUSCITATION COMPONENTS: RATIOS BY AGE

Ventilation Mode	Respirations	Chest Compressions	Notes
Bag-mask ventilation	• 15 chest compressions • 2 rescue breaths after each • If only 1 rescuer, 2 rescue breaths after each 30 compressions	100-120 per minute	Decompress stomach to alleviate gastric inflation.

Endotracheal intubation	8-10 rescue breaths per minute	100-120 per minute	• Do not interrupt compressions during ventilation. • Confirm appropriate positioning of the endotracheal tube.
Neonates	3:1 ratio of compressions to rescue breaths	90 compressions per minute	

Chest compressions generate aortic pressure and coronary perfusion pressure. When compressions cease, the aortic diastolic pressure rapidly falls, dropping coronary perfusion pressure. Increasing the ratio of compressions to ventilations minimizes these interruptions, thus increasing coronary perfusion pressure (blood flow). The benefits of positive pressure ventilation (increased arterial transport of oxygen and carbon dioxide elimination) must be balanced against the adverse consequence of impeding venous return and cardiac output. This can be achieved by applying the recommended compression-to-ventilation ratios (**Table 3-1**).

2. Rate of Ventilation

The recommended respiratory rate during pediatric CPR is 8 to 10 breaths per minute in synchrony with compressions when the patient is not intubated, and 8 to 10 breaths per minute asynchronously (not coordinated with compressions) in the intubated patient. A common error during the stress of a pediatric cardiac arrest is for providers to deliver ventilation at rates or depths that are substantially higher than recommended. As noted earlier, high ventilation rates increase intrathoracic pressure, impede venous return and coronary artery blood flow, and can result in a lower likelihood of ROSC. Therefore, it is important to monitor the rate of ventilation.

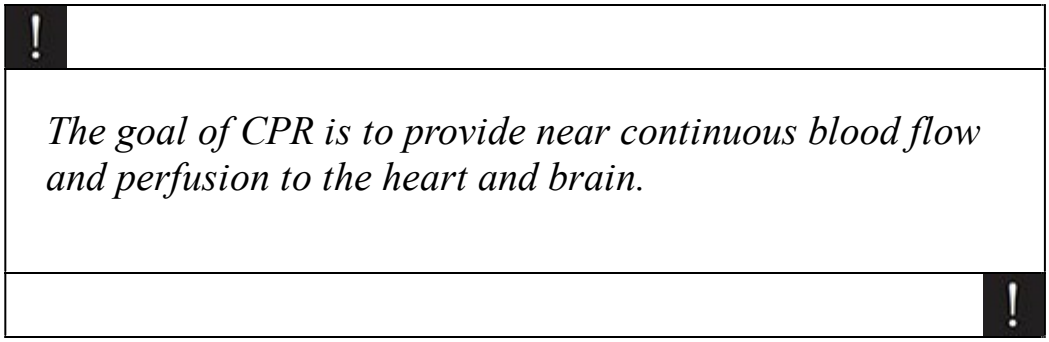
D. Oxygen Management and Monitoring

Pediatric resuscitation for cardiac arrest, beyond the delivery room, should be initiated with 100% oxygen, when available. While studies in neonatal and animal resuscitation have demonstrated evidence of delayed first breath and poor neurologic outcomes following resuscitation with 100% oxygen (as opposed to room air), similar data do not exist in the pediatric group. The AHA Pediatric Advanced Life Support (PALS) 2015 guidelines continue to recommend the use of 100% oxygen whenever chest compressions are provided, and to wean the oxygen concentration once there is a return of spontaneous circulation. The fraction of inspired oxygen administered to a child with ROSC should be actively titrated to avoid both hypoxia and hyperoxia in the post-resuscitation period. Current guidelines suggest that the fraction of inspired oxygen be decreased to the lowest possible level while maintaining oxygen saturation levels between 94% and 99% (ie, less than 100%). This recommendation is based on the observation that, in the presence of an oxyhemoglobin saturation of 100%, the Pao_2 may vary from 80 mm Hg to >500 mm Hg.

VI. QUALITY OF CARDIOPULMONARY RESUSCITATION

A. Monitoring

The goal of CPR is to provide near continuous blood flow and perfusion to vital organs. Clinical evidence of good-quality CPR during cardiac arrest can include return of movement, persistence of gasping, and eye opening.



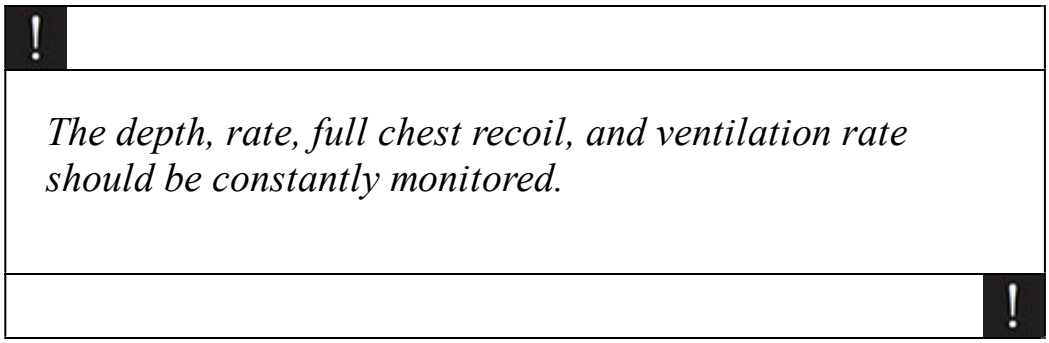
Animal and human studies have demonstrated that changes in cardiac output during resuscitation from shock and cardiac arrest are associated with exhaled or end-tidal carbon dioxide ($ETCO_2$). Continuous monitoring of $ETCO_2$ waveforms may be useful as a marker of pulmonary blood flow during CPR. An abrupt or sustained marked increase in $ETCO_2$ to >40 mm Hg can be an early indicator of ROSC, avoiding the need to interrupt chest compressions. Multiple studies in adults have shown that an $ETCO_2$ <15 mm Hg during CPR is associated with a decreased likelihood of ROSC.

If CPR is in progress on a patient with continuous $ETCO_2$ capnography waveform monitoring in place, an $ETCO_2$ of <15 mm Hg should prompt the rescuer to increase the quality of chest compressions until the $ETCO_2$ is consistently >15 mm Hg. Attention should also be paid to ventilation when $ETCO_2$ <15 mm Hg, as inappropriate hyperventilation during CPR may lower $ETCO_2$.

The utility of $ETCO_2$ for predicting outcomes, or for determining appropriate timing of terminating efforts, is limited by the lack of definitive $ETCO_2$ target cutoff values in cardiac arrest patients.

B. Quality of CPR Monitoring

The depth, rate, full chest recoil, and continuation of chest compressions, as well as the ventilation rate, should be constantly monitored, with or without adjuncts. Because poor-quality CPR is associated with worse outcome and reduces the likelihood of defibrillation success, rescuers should focus on the quality of their efforts. Automated feedback devices to improve CPR quality and compliance with guidelines are available.



1. Vascular Access

Rapid vascular access is essential in patients in cardiac arrest or who are at imminent risk of arrest. Obtaining IV access can be difficult in the arrested patient. Intraosseous (IO) access has been recognized as safe and quickly effective in children and adults, and the frequency of its complications is negligibly low. Current recommendations state that IO access may be considered as the initial approach to vascular access in the arrested pediatric patient (**Appendix 2**). Multiple mechanical devices designed to assist with IO access have been shown to be effective and easy to use in pediatric patients.

Although some resuscitation medications (eg, epinephrine, atropine) have been shown to reach the central circulation when instilled through the endotracheal tube into the tracheobronchial tree, their absorption is unreliable. Widespread acceptance of IO access has obviated the need for endotracheal administration, and this approach is no longer recommended.

!
Place an IO needle if IV access is not immediately achievable.
!

VII. PEDIATRIC VENTRICULAR FIBRILLATION AND DEFIBRILLATION

A. Dosing Recommendations

Defibrillation is necessary for successful resuscitation from VF cardiac arrest (**Appendix 9**). When prompt defibrillation is provided soon after the induction of VF in a cardiac catheterization laboratory, the success and survival rates approach 100%. In general, the mortality rate increases 5% to 10% per minute of delay to defibrillation. Because pediatric cardiac arrests are commonly due to progressive asphyxia or shock (or both), the initial focus of treatment is generally prompt CPR. However, in sudden VF cardiac arrest in children, prompt defibrillation is the treatment of choice. Thus, the emphasis on early CPR versus early defibrillation must be balanced against the increasing evidence that VF in children is not rare, that outcomes from arrhythmogenic VF arrests are superior to those from other types of cardiac arrests, and that early rhythm recognition is necessary for optimal care.

!
Do not delay defibrillation to establish an advanced airway or vascular access.

Because of the increasing awareness that "shockable" rhythms are not uncommon in children (eg, approximately 5% to 15% of initial cardiac arrest rhythms), greater attention has been focused on the dosage for pediatric defibrillation. The recommended initial shock dose is 2 to 4 J/kg, followed by immediate resumption of chest compressions. For refractory VF, it is reasonable to increase the dose to 4 J/kg. For subsequent energy levels, a dose of 4 J/kg may be reasonable and higher energy levels may be considered, although they should not exceed 10 J/kg or the maximum adult dose.

In sudden VF cardiac arrest in children, prompt defibrillation is the treatment of choice.

B. Automated External Defibrillators

Automated external defibrillators (AEDs) with a dose attenuator (which decreases the amount of power the patient receives) are recommended for children younger than 8 years (<25 kg). For children <1 year of age, a manual defibrillator is preferred. In the event a dose attenuator (ages 1-8 years) or a manual defibrillator (<1 year) is not available, an AED still may be used, because untreated VF is fatal without a shock.

Large electrode pads should be used for children according to the manufacturer's recommendations. The use of self-adhesive (hands-free) pads is preferred when possible.

VIII. MEDICATIONS

A. Epinephrine (Adrenaline)

During CPR, epinephrine's α-adrenergic effect increases systemic vascular resistance, increasing diastolic blood pressure, which in turn increases the coronary perfusion pressure and blood flow and increases the likelihood of ROSC (**Table 3-2**). Epinephrine also increases cerebral blood flow

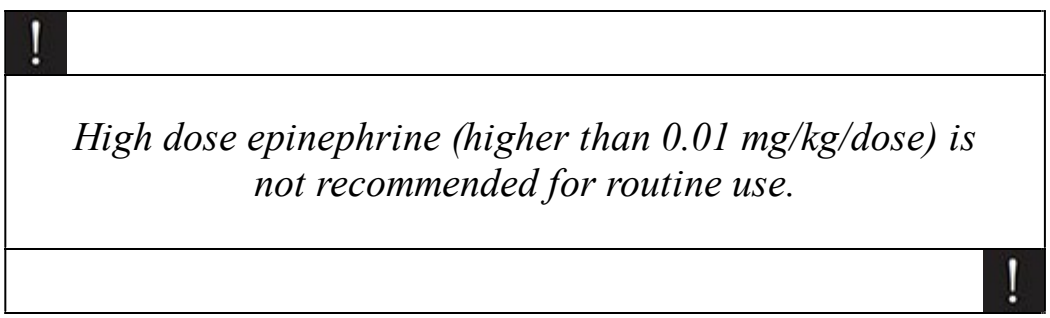
during CPR because peripheral vasoconstriction directs a greater proportion of flow to the cerebral circulation. The β-adrenergic effect increases myocardial contractility and heart rate and relaxes smooth muscle in the skeletal muscle vascular bed and bronchi, although this effect is of less importance. Epinephrine also changes the character of VF (ie, higher amplitude, more "coarse"), increasing the likelihood of successful defibrillation.

Table 3-2 DRUG DOSING

Drug	Dosing
Epinephrine (adrenaline)	Pulseless arrest • 0.01 mg/kg (0.1 mL/kg) of 1:10,000 concentration IV/IO, repeat every 3-5 min (maximum 1 mg/dose)
Amiodarone	Pulseless arrest • 5 mg/kg IV/IO bolus (maximum 300 mg) SVT/VT (with pulse): • 5 mg/kg IV/IO over 20-60 min (maximum 300 mg)
Lidocaine	VF/pulseless VT • 1 mg/kg IV/IO bolus (maximum cumulative dose 3 mg/kg) loading dose, followed by infusion of 20-50 µg/kg/min
Vasopressin	Pulseless arrest (may be effective in refractory cardiac arrest when standard therapy fails) • 0.4-1 U/kg bolus (maximum 40 U)
Atropine	Antidotal therapy
Sodium bicarbonate	Hyperkalemia (or documented severe metabolic acidosis) or some toxidromes (eg, tricyclic antidepressant poisoning) • 1 mEq/kg IV/IO bolus
Calcium chloride (central venous line only)	Documented severe hypocalcemia or hyperkalemia with arrest • 20 mg/kg (0.2 mL/kg) IV/IO (maximum dose 2,000 mg) slow push for hypocalcemia
Calcium gluconate	60-100 mg/kg (0.6-1.0 mg/kg) (maximum 3,000 mg) slow IV/IO push

IO, intraosseous; SVT, supraventricular tachycardia; VT, ventricular tachycardia; VF, ventricular fibrillation

Prospective and retrospective studies indicate that use of high-dose epinephrine in adults or children (0.05-0.2 mg/kg) does not improve survival and may be associated with a worse neurologic outcome. Thus, it is not recommended for routine use during CPR.



B. Amiodarone or Lidocaine

Amiodarone and lidocaine are recommended as potential first-line medical therapy for defibrillation-

resistant VF/pulseless ventricular tachycardia (pVT) in adults. In adult studies comparing amiodarone and lidocaine some reported improvement in ROSC and survival to hospital admission with amiodarone, but no survival to discharge benefit.

No pediatric data conclusively support the efficacy of either amiodarone or lidocaine in cardiac arrest due to VF/pVT. Pediatric case reports have demonstrated that amiodarone is effective in terminating life-threatening ventricular dysrhythmias, not cardiac arrest. A recent, multi-institution registry of inpatient pediatric cardiac arrest demonstrated that, compared with amiodarone, lidocaine was associated with higher rates of ROSC and 24-hour survival, but neither lidocaine nor amiodarone was associated with improved survival to hospital discharge. Pediatric guidelines for antiarrhythmic therapy are extrapolated from adult data, and either amiodarone (5 mg/kg) or lidocaine (1 mg/kg) may be used.

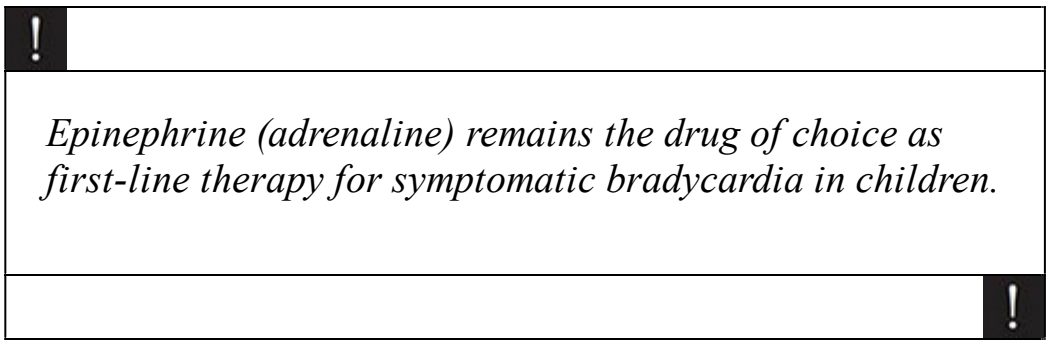
C. Vasopressin

Vasopressin, a non-catecholamine vasopressor, has been shown to be an effective adjunctive and initial vasoactive medication in adult cardiac arrest. Studies on its impact in pediatric cardiac arrest are limited. Data are equivocal, with one retrospective study showing lower rates of ROSC with vasopressin, while multiple case reports have shown that vasopressin resulted in successful ROSC in adult and pediatric cardiac arrests refractory to standard therapy. Published dosing for children is 0.4 to 1 U/kg. Evidence on vasopressin in pediatric cardiac arrest is insufficient to recommend for or against its use.

D. Other Agents

1. Atropine

Evidence is insufficient to support or refute the use of atropine in pediatric cardiac arrest. It may be used for symptomatic bradycardia known to be due to increased vagal tone or drug or environmental intoxications marked by cholinergic excess (eg, organophosphates, sarin, VX, or other nerve agents). Epinephrine remains the drug of choice as first-line therapy for symptomatic bradycardia in children.



2. Bicarbonate

Sodium bicarbonate is not recommended for routine use in pediatric cardiac arrest. Although no randomized trials exist, several studies suggest an association between bicarbonate use and

decreased survival, even when controlling for other potentially confounding factors. Exceptions to this recommendation would include cases of cardiac arrest due to special resuscitation circumstances where sodium bicarbonate therapy is part of standard and specific treatment (eg, tricyclic antidepressant overdose resulting in ventricular dysrhythmia, life-threatening hyperkalemia, severe pulmonary hypertension with acidosis).

3. Calcium

Data do not support routine use of calcium salts in cardiac arrest. Two controlled analyses of multihospital data demonstrated significantly decreased survival to hospital discharge among children receiving calcium salts during CPR. Recommendations for their use during CPR are limited to special resuscitation circumstances, such as documented hypocalcemia, hyperkalemia, or known or suspected intoxication with calcium channel blockers.

4. Magnesium

Magnesium is the initial drug recommended for torsades de pointes in adults. No specific recommendations exist for its use in pediatric cardiac arrest. Magnesium should be administered for documented hypomagnesemia. Because magnesium is a vasodilator, hypotension can occur during IV administration.

5. Glucose

Routine use of glucose-containing fluids during resuscitation is not recommended. In cases of documented hypoglycemia, glucose should be administered in doses of 0.5 g/kg (calculated rapidly by the “rule of 50”: 1 mL/kg 50% dextrose, or 2 mL/kg 25% dextrose, or 5 mL/kg 10% dextrose). The empiric administration of glucose is not recommended for cardiac arrest in children.

IX. SPECIFIC RHYTHMS

Case Study

A 7-year-old boy is struck in the chest by a baseball. Moments later, he collapses on the field. His coach comes to his side to find him unresponsive and apneic; CPR is begun and the emergency medical service is called. An AED is applied and a shock is recommended. Following one shock, CPR is resumed.

Detection

– After 2 minutes of CPR, what essential assessment(s) should be performed?

– A faint brachial pulse is appreciated; the boy starts to breathe spontaneously and opens his eyes.

Intervention

– What is the most appropriate immediate action now?

Reassessment

– If the single shock had been unsuccessful, what would be the next action?

– If the boy continued to be pulseless, unresponsive, and apneic, what medications would be recommended?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

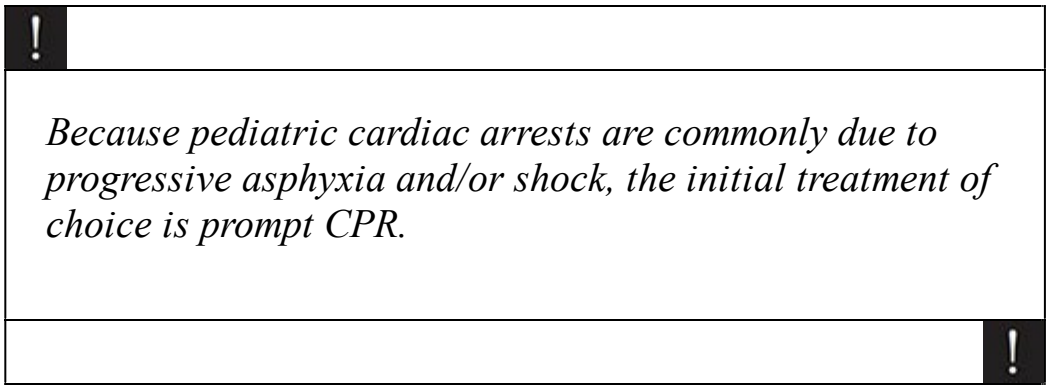
– Who is to do what and when?

This is a case of commotio cordis, where sudden low-energy blunt chest trauma can cause ventricular fibrillation VF in otherwise healthy young individuals. If the chest wall impact occurs during a narrow vulnerable window of ventricular repolarization, the generated premature ventricular impulse can lead to VF and cardiac arrest.

A. VF/pVT (Shockable Rhythms)

VF is an uncommon, but not rare, electrocardiographic rhythm during out-of-hospital pediatric cardiac arrests (**Appendix 9**). Its incidence varies by setting and age. In special circumstances, such as commotio cordis, tricyclic antidepressant overdose, cardiomyopathy, postcardiac surgery, and prolonged QT syndromes, VF is a more likely rhythm during cardiac arrest. Commotio cordis or mechanically initiated VF due to relatively low-energy chest wall impact during a narrow window of repolarization (10-30 millisecond before the T-wave peak in swine models) is reported predominantly in children age 4 to 16 years. Out-of-hospital VF cardiac arrest is uncommon in infants, but occurs more frequently in children and adolescents. Although VF is often associated with underlying heart disease and generally considered the "immediate cause" of cardiac arrest (ie, an arrhythmogenic arrest), "subsequent" VF can also occur during resuscitation efforts (ie, after an initial rhythm of asystole or pulseless electrical activity [PEA]). Traditionally, VF and ventricular tachycardia (VT) have been considered "good" cardiac arrest rhythms, resulting in better outcomes than asystole and PEA. Survival to discharge is much more common among children with an initial shockable rhythm than among those with shockable rhythms occurring later during the resuscitation. Data suggest that outcomes after initial VF/VT are "good," but outcomes after subsequent VF/VT are substantially worse, even compared to initial asystole/PEA without subsequent VF/VT.

Defibrillation (defined as termination of VF by administering a controlled electric shock) is necessary for successful resuscitation from VF cardiac arrest. The termination of fibrillation can result in asystole, PEA, or a perfusing rhythm. The goal of defibrillation is return of an organized electrical rhythm with pulse. When prompt defibrillation is provided soon after the induction of VF in a cardiac catheterization laboratory, the rates of successful defibrillation and survival approach 100%. When AEDs are used within 3 minutes of adult-witnessed VF, long-term survival can occur in more than 70%. Early and effective, near-continuous chest compressions can attenuate the increase in the mortality rate with delayed defibrillation. Because pediatric cardiac arrests are commonly due to progressive asphyxia and/or shock, the initial treatment of choice is prompt CPR. Therefore, rhythm recognition is emphasized less than in adult cardiac arrests. The earlier that VF can be diagnosed and treated, the higher the success.


!
Because pediatric cardiac arrests are commonly due to progressive asphyxia and/or shock, the initial treatment of choice is prompt CPR.
!

B. Bradycardia: Symptom of Hypoxia and/or Hypoperfusion

Case Study

A 2-year-old girl is brought to an emergency department by emergency medical services after a bystander found her unresponsive in a parking lot. En route to the hospital, she was noted to be taking 6 breaths/min, so she received assisted ventilation. On arrival, she has a narrow complex rhythm at a rate of 38 beats/min with faint pulse, and an undetectable blood pressure.

Detection

– What are the possible causes of this scenario?

Intervention

– What is the most important next step?

Reassessment

– After interventions are performed, what is the rhythm?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

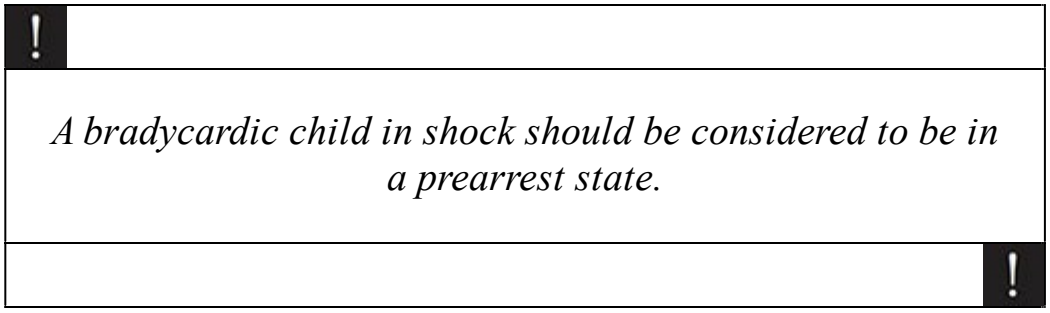
Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Neonates, infants, and children are primarily dependent on heart rate for maintenance of cardiac output. They rarely have heart block or a primary cardiac reason for bradycardia. Most frequently bradycardia in infants and children is a manifestation of hypoxia and hypoperfusion. The ability to augment stroke volume to increase cardiac output is limited, and physiologic or pathophysiologic states leading to an increase in cardiac output have tachycardia as their hallmark. Conversely, illnesses or injuries resulting in negative chronotropic actions (eg, heart block, toxicity due to β-blockers or calcium channel blockers) tend to result in more profound shock and hypoperfusion in children than in adults.

Bradycardia with hypoperfusion (with pulse) is a common hemodynamic state for critically ill children during the prearrest phase. Animal models of asphyxia have demonstrated a predictable hemodynamic progression from tachycardia to bradycardia with hypotension, followed by PEA and asystole. Earlier CPR in this continuum is associated with more favorable outcomes. Given that most children in cardiac arrest were experiencing either respiratory insufficiency and/or circulatory insufficiency prior to the onset of pulselessness, a bradycardic child in shock should be considered to be in a prearrest state. Multiple reversible causes are possible, but immediate support of cardiovascular status is essential. Neonatal resuscitation algorithms have recommended escalation of respiratory and cardiac support for the neonate whose heart rate is <60 beats/min, including the provision of chest compressions if bradycardia does not resolve with effective ventilation and oxygenation. Multiple studies have shown that CPR is frequently initiated for hospitalized children when they are bradycardic and hypoperfused, and that chest compressions initiated before the onset of pulselessness are associated with improved survival. Current AHA guidelines recommend considering immediate chest compressions for a child with a heart rate <60 beats/min and signs and symptoms of hypoperfusion.



C. Supraventricular Tachycardia

Case Study

A 3-month-old infant is brought to the emergency department by a parent who reports that she was well until 1 hour ago. Since then, she has become progressively lethargic, and she is now unresponsive. Initial assessment reveals irregular, gasping respirations, and a very weak, rapid, barely palpable pulse. An electrocardiogram shows a narrow complex ECG rhythm with a heart rate of 260 beats/minute. Bag-mask ventilation is initiated with good chest rise, and a pediatric code is announced.

Detection

– What are the key aspects of the initial assessment of this infant?

– What is the likely diagnosis?

Intervention

– What is the most important next step?

Reassessment

– What are the next management steps?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Supraventricular tachycardia, a relatively common arrhythmia in infants and children, may be associated with severe circulatory compromise or even cardiac arrest. Therapy for this arrhythmia should be based on the child's hemodynamic status. In the case of hemodynamically unstable supraventricular tachycardia, immediate cardioversion with 0.5 J/kg should be performed, increasing to 1 J/kg if necessary. Adenosine may also be considered first-line therapy, although IV access is required. In the unstable patient, cardioversion should not be delayed by attempts to achieve vascular access. Appropriate sedation and analgesia may be needed for conscious children before cardioversion is performed.

The initial dose of adenosine is 0.1 mg/kg given as a rapid IV/IO bolus. Central venous administration is preferable because the drug is rapidly metabolized by red blood cell adenosine

deaminase, with a half-life of less than 10 seconds. When the drug is given, the catheter should be immediately and rapidly flushed with at least 10 mL of saline.

D. Pulseless Electrical Activity

Case Study

A 5-year-old girl with necrotizing pneumonia and septic shock is being managed in the pediatric ICU with positive pressure ventilation and an epinephrine infusion (0.3 μg/kg/min). She has an acceptable blood pressure, a pulse oximeter reading in the mid-90% range, and $ETCO_2$ (exhaled) of 40 mm Hg. Acutely, her pulse oximetry reading falls to 70% and then loses its waveform, the $ETCO_2$ drops to zero, and the waveform on her arterial catheter becomes flat. Her ECG shows a narrow complex rhythm with a rate of 40 beats/min, and she becomes unconscious.

Detection

– What essential assessments should be performed?.

Intervention

– What is the most important immediate step?

– CPR is immediately initiated. Assessment of airway and breathing reveals no breath sounds on the right.

– A needle is inserted in the second intercostal space in the mid-clavicular line with prompt return of spontaneous circulation.

Reassessment

– What are the next management steps?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

PEA is defined as organized ECG activity, excluding ventricular tachycardia and fibrillation, without clinical evidence of a palpable pulse or myocardial contractions. The most common causes of PEA can be remembered as the *6 H's* and the *6 T's*, as summarized in **Table 3-3**. All of these causes of PEA are potentially reversible; therefore, prompt efforts should be made to identify and correct them during the CPR. While such efforts are under way, epinephrine (10 μg/kg) may be administered every 5 minutes. When the cause of PEA is unknown and the patient does not respond to medications, consider administering a fluid bolus and inserting catheters into the pleural space to rule out pneumothorax, and into the pericardial space to rule out cardiac tamponade. A rapid bedside ultrasound exam may be helpful in evaluating for rapid diagnosis (eg, pericardial effusion, pneumothorax, pulmonary embolus).

Table 3-3 POTENTIALLY REVERSIBLE FACTORS IN CARDIAC ARREST

The 6 H's	**The 6 T's**
Hypovolemia	Tension pneumothorax
Hypoxia	Tamponade, cardiac
Hydrogen ion (acidosis)	Toxins
Hypoglycemia	Thrombosis, pulmonary
Hypo-/hyperkalemia	Thrombosis, coronary
Hypothermia	Trauma

X. SPECIAL RESUSCITATION CIRCUMSTANCES

A. Newborn

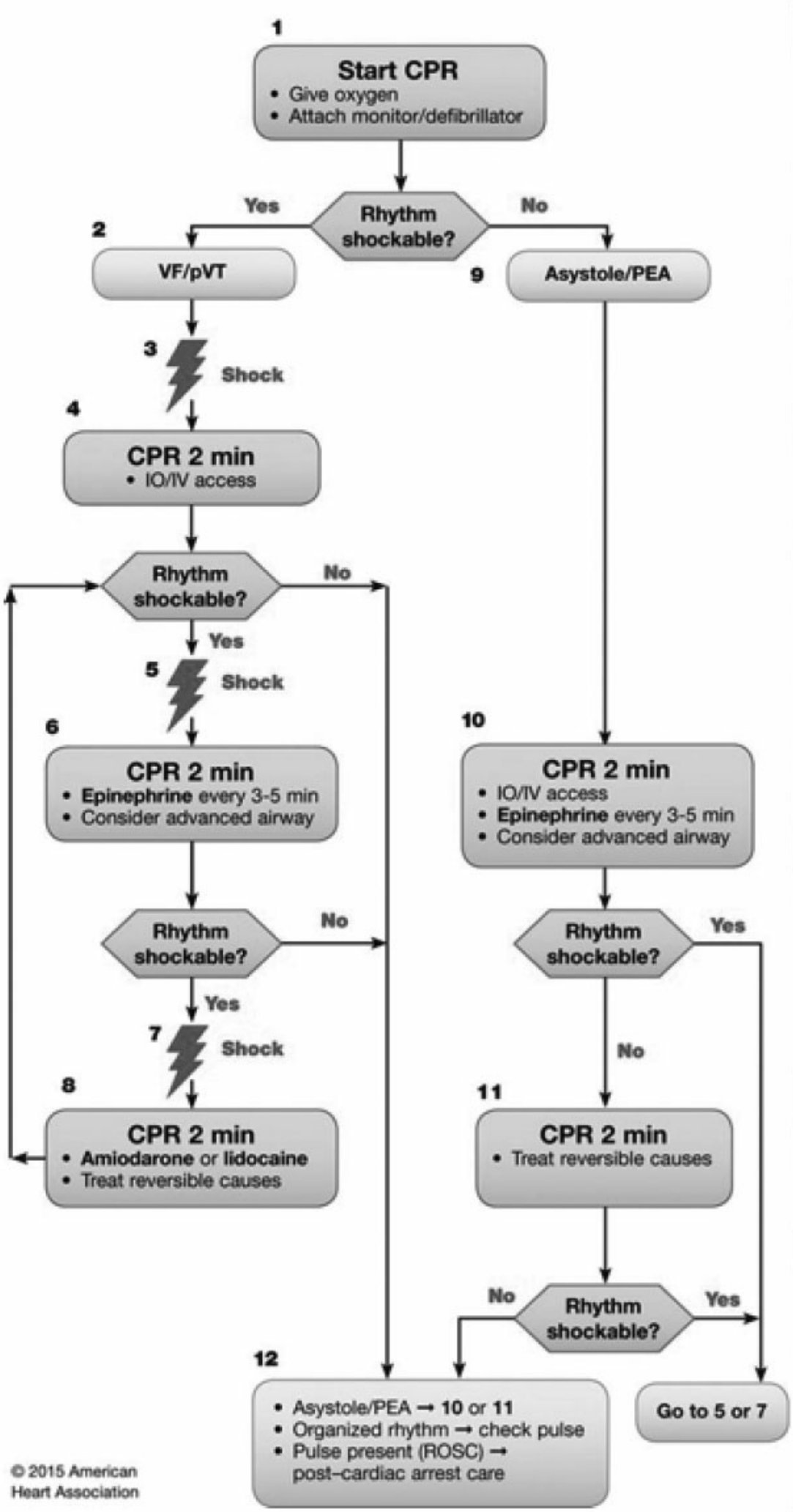
Worldwide, approximately 4 million newborns suffer from birth asphyxia, resulting in approximately 1 million deaths and another million with neurologic sequelae. Neonatal resuscitation research has focused on how to effectively resuscitate the newborn and decrease the risk of neurologic injury and death. The focus is on clearing and opening the airway, initiating the first few breaths effectively to resuscitate the compromised neonate, and adding positive pressure or continuous positive airway pressure as needed. If the heart rate drops below 100 beats/min, assisted ventilation should be provided. If the rate is <60 beats/min despite 30 seconds of ventilation with the addition of supplemental oxygen, chest compressions should be initiated and intubation should be considered. CPR should be performed utilizing a ratio of 3:1, synchronously targeting 90 compressions/min and 30 breaths/min (eg, 120 events/min). Recent neonatal guidelines focus on using room air resuscitation to avoid hyperoxia. Room air resuscitation has been studied extensively and improves the time to first spontaneous breath and the mortality rate. Recommendations are that it should be initiated and, if bradycardia is <60 beats/min and persists for 90 seconds, 100% oxygen should be titrated until recovery of a normal heart rate, and thereafter guided by oximetry to avoid hyperoxia.

B. Congenital Heart Disease/Pulmonary Hypertension

Standard prearrest and arrest resuscitation should be provided to children with congenital heart disease. Infants in the first few weeks of life who present hypoperfused or in cardiac arrest should be evaluated for the common causes of pediatric arrest, but ductal-dependent congenital heart disease should also be considered.

Patients with single-ventricle physiology and pulmonary blood flow dependent on a systemic-to-pulmonary artery shunt who present with persistent hypoxemia or full arrest may have evolving shunt thrombosis. They may be treated with systemic heparinization to prevent ongoing thrombosis, and systemic vascular resistance can be increased (fluid boluses and phenylephrine) to promote pulmonary blood flow. Patients with known pulmonary hypertension and associated right ventricular dysfunction are at high risk for cardiac arrest. Increases in intrathoracic pressure due to positive pressure ventilation can result in inadequate cardiac output and arrest. During cardiac arrest, it may helpful to treat acidosis and administer IV fluids to provide preload to the systemic ventricle. Even in these special circumstances, clinicians should follow standard pediatric advanced life support guidelines for pediatric resuscitation (**Figure 3-1**). Early consultation with a pediatric cardiologist or cardiac intensivist may be critical to the treatment of these patients.

Figure 3-1. American Heart Association Pediatric Cardiac Arrest Algorithm



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CPR Quality

- Push hard (≥⅓ of anteroposterior diameter of chest) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 15:2 compression-ventilation ratio.

Shock Energy for Defibrillation

First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose

Drug Therapy

- **Epinephrine IO/IV dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes.
  If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.
- **Lidocaine IO/IV dose:** Initial: 1 mg/kg loading dose. Maintenance: 20-50 mcg/kg per minute infusion (repeat bolus dose if infusion initiated >15 minutes after initial bolus therapy).

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypoglycemia
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; VT, ventricular tachycardia; PEA, pulseless electrical activity; IO, intraosseous; ET, endotracheal

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C. Anaphylaxis

Case Study

Within minutes of eating a cookie, a 12-year-old girl with a known nut allergy developed dyspnea, lip swelling, hives, flushing, and dizziness; she complained of an "impending sense of doom." She utilized her epinephrine auto-injector, and emergency medical services was called. When they arrived 10 minutes later, she was cyanotic, in severe distress, and minimally responsive. Tracheal intubation was performed emergently in the field. While en route to the hospital, she became bradycardic and hypoxic and progressed to cardiopulmonary arrest.

Detection

– What is the patient's current clinical condition?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– What is the patient's response to treatment?

– How does management of this patient differ from that of other cardiac arrest patients?

– What are the next management steps?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Anaphylaxis is a severe systemic allergic reaction that can be reversed with rapid and adequate treatment, but it can be fatal without appropriate intervention. Symptoms include tachycardia,

flushing, diarrhea, bronchospasm, angioedema, and urticaria; the patient's condition can progress to profound hypotension and cardiovascular collapse. Vasodilation and increased capillary permeability cause relative intravascular depletion and hypotension with systemic hypoperfusion that can progress to cardiac arrest. Standard basic life support and advanced cardiac life support should be utilized for treatment of cardiac arrest, but adjunctive medications should be administered for the specific management of anaphylaxis.

A healthcare provider with advanced airway skills may be needed early as lip, pharyngeal, and glottic edema are common and can impede attainment of a secure airway. Intramuscular epinephrine should be administered immediately and can be repeated. Fluid resuscitation should be provided with repeated crystalloid solution boluses of 20 mL/kg to treat the hypotension. Persistent hypotension should be managed with continued fluid resuscitation and an epinephrine infusion. To further alleviate the ongoing symptoms, patients should also receive IV steroids, antihistamines (diphenhydramine, H_2 blocker), and inhaled β-agonists (racemic epinephrine or albuterol).

D. Trauma

Cardiac arrest in trauma, particularly blunt trauma, is associated with high mortality rates, but reversible causes of arrest should be sought during CPR and treated. Basic and advanced life support should be instituted immediately. Because of the potential for neck injury, a jaw thrust with cervical spine immobilization should be employed to open the airway. For the trauma patient who progresses to cardiac arrest, the likely causes are hypovolemia from ongoing bleeding, hypothermia from exposure, tamponade from chest trauma, or hemothorax or pneumothorax. Aggressive volume resuscitation with crystalloid and/or blood products should be instituted with the goal of achieving normovolemia, and a core temperature should be taken and managed. Breath sounds, radiographs and bedside ultrasound should be evaluated for evidence of hemothorax or pneumothorax; if present, chest tube placement should be considered. Cardiac tamponade is another potentially reversible cause of traumatic cardiac arrest that can be treated with pericardiocentesis or emergent thoracotomy. Spinal cord injury can result in hypotensive shock without reflex tachycardia. Specific treatment should focus on increasing vasomotor tone and supplementing chronotropic action and vasoconstriction while instituting volume resuscitation.

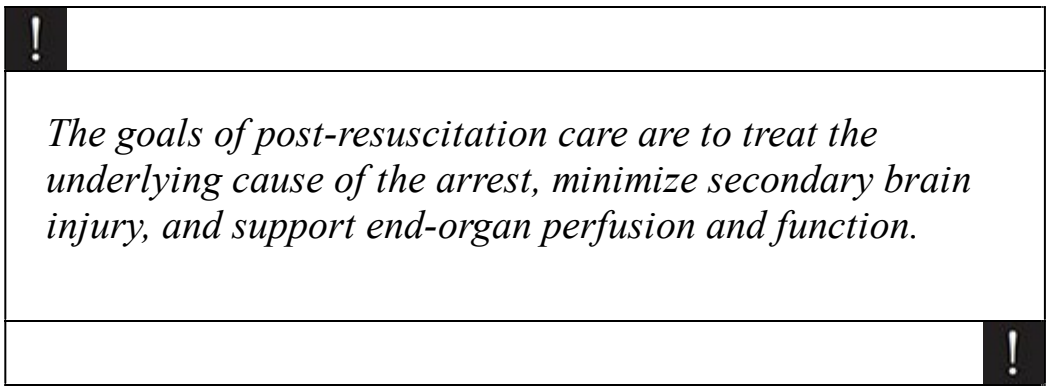
XI. POST-RESUSCITATION MANAGEMENT

The goals of post-resuscitation care are to diagnose and treat the underlying cause of the arrest, minimize secondary brain injury, and support end-organ perfusion and function. Aggressive post-resuscitative care may improve survival and neurologic outcomes. Specific attention should be paid to oxygenation, ventilation, temperature control, seizure control, glucose and electrolyte levels, and hemodynamics, with the goal of matching appropriate substrate delivery to critical end-organ tissue demands (**Table 3-4**).

Table 3-4	CARDIAC ARREST TREATMENT TIMELINE

First 5 minutes	First 3 hours
• Activate emergency code team, assign team member roles • Initiate high-quality CPR • Identify potentially reversible causes	• Prevent or treat hypotension • Prevent or treat fever • Maintain normoxia, normal ventilation, and electrolyte/glucose levels • Consult other specialties, as needed
First 30 minutes	**First 48 hours**
• If ROSC achieved: determine location of definitive care; check temperature, pulse oximetry, lab results; monitor and treat hypotension • If resuscitation ongoing: determine if resuscitation should continue based on multiple criteria	• Prevent or treat hypotension • Prevent or treat fever • Monitor for seizures and treat status epilepticus • Manage temperature, oxygenation, ventilation, electrolytes

CPR, cardiopulmonary resuscitation; ROSC, return of spontaneous circulation


The goals of post-resuscitation care are to treat the underlying cause of the arrest, minimize secondary brain injury, and support end-organ perfusion and function.

A. Oxygenation

Following ROSC, both persistent hypoxemia (low Pao_2) and hyperoxemia (elevated Pao_2) have been associated with poor neurologic outcomes and concerns that hyperoxia may worsen oxidative injury during reperfusion. Therefore, one goal of post-resuscitative care is to deliver adequate oxygen to the patient while minimizing the risk of ongoing oxidative stress. Many patients do not have arterial monitoring early after arrest, but if a patient's saturation is 100%, practitioners should wean the supplemental oxygen to a saturation goal between 94% and 99%.

B. Ventilation

Arterial CO_2 levels directly impact cerebral vascular tone in healthy patients. Following cardiac arrest, cerebral edema may occur and titration of arterial CO_2 may impact blood flow. Hyperventilating patients to an arterial CO_2 <35 mm Hg can result in cerebral vasoconstriction, hypoperfusion of the already injured brain, and increased intrathoracic pressure, thus limiting cardiac output. Likewise, hypoventilation can lead to cerebral vasodilation and increase already elevated intracranial pressure. Although an individual's physiology is variable and intracranial pressure and cerebral blood flow monitoring are not standard after cardiac arrest, clinicians should maintain a normal arterial CO_2 between 35 and 45 mm Hg. If an arterial line is not in place, an $ETCO_2$ monitor can be used; however, it should correlate with an arterial blood gas because dead space ventilation is

variable among patients and the difference between arterial and $ETCO_2$ also is variable.

C. Cardiovascular Support

Postarrest myocardial stunning and hypotensive shock are common after successful resuscitation in children and adults. These are generally reversible states among long-term survivors. Postarrest myocardial stunning is pathophysiologically similar to sepsis-related and post-cardiopulmonary bypass myocardial dysfunctions, including increases in inflammatory mediator and nitric oxide production. While the optimal management of postcardiac arrest hypotension and myocardial dysfunction has not been established, adult data suggest that early hypotension in the 6 hours after ROSC is associated with increased mortality; therefore, aggressive hemodynamic support may improve outcomes.

Initially, fluid resuscitation should be provided for hypotension. The volume should be titrated to optimize the central venous pressure if it is being monitored. Inotropic and/or vasopressor support should be provided for hypotension caused by myocardial dysfunction or vasodilation. Monitoring central venous pressure, central venous oxygen saturation, lactate levels, and urine output may help guide the effectiveness of therapies. General critical care principles suggest that appropriate therapeutic goals are adequate blood pressures; oxygen delivery; and myocardial, cerebral, and systemic blood flows. Reasonable interventions for vasodilatory shock with low central venous pressure include fluid resuscitation and vasoactive infusions. Appropriate considerations for left ventricular myocardial dysfunction include euvolemia, inotropic infusions, and afterload reduction. Echocardiography may contribute adjunctive data to guide therapies and may be repeated as needed.

D. Temperature

Hyperthermia following cardiac arrest is common in children. Animal and human data show that fever following brain injury is associated with worse outcomes; therefore, fever should be aggressively prevented and treated following cardiac arrest. Induced hypothermia (32°C-34°C [89.6°F-93.2°F]) following witnessed adult VF cardiac arrest improves survival and neurologic outcomes and may be considered for children who remain comatose after pediatric cardiac arrest; however, prospective pediatric data have no proven efficacy when applied to deeply comatose children who survived in- or out-of-hospital arrest. Observational data on pediatric patients resuscitated from cardiac arrest suggest that ICU duration of stay, neurologic outcomes, and mortality are unchanged with the use of therapeutic hypothermia. One small study of therapeutic hypothermia in survivors of pediatric asphyxia cardiac arrest showed an improvement in mortality at hospital discharge, with no difference in neurologic outcomes. Two large multi-institutional, prospective, randomized studies of pediatric comatose survivors of cardiac arrest (therapeutic hypothermia after pediatric cardiac arrest out-of-hospital and in-hospital) found no difference in survival with good functional outcome at 1 year and no additional complications. Investigators compared therapeutic hypothermia (32°C-34°C [89.6°F-93.2°F]) to therapeutic (active) normothermia (36°C-37.5°C [96.8°F-99.5°F]). It is reasonable either to maintain 5 days of continuous normothermia or to maintain 2 days of initial continuous hypothermia followed by 3 days of continuous normothermia. Thus, continuous measurement of temperature during this period is recommended, and fever (temperature

≥38°C [≥100.4°F]) should be aggressively treated.

Following ROSC, clinicians should continuously measure the patient's core temperature (rectal, bladder, or esophageal). It is not uncommon for small children who have had a prolonged resuscitation to be hypothermic. Clinicians should discuss temperature goals with a tertiary care center. At a minimum, hyperthermia (temperature >38°C [>100.4°F]) should be prevented with antipyretics and a cooling blanket. Similarly, hypothermia (≤32°C [≤89.6°F]) should be corrected as deep hypothermia can lead to arrhythmias and repeat cardiac arrest.

E. Glucose

Hyperglycemia following adult cardiac arrest is associated with worse neurologic outcomes, as is hypoglycemia. Data for evidence-based titration of specific endpoints in children are not available. Therefore, clinicians should frequently monitor blood glucose levels and aim for normoglycemia.

F. Seizures

Seizures following cardiac arrest are common and often are undetectable without electroencephalography (EEG). Such seizures (clinical and subclinical) are associated with worse neurologic outcomes, although it is unclear whether they are the result of severe brain injury, cause severe brain injury, or both. Clinical convulsions should be aggressively treated and evaluation for correctable causes should be sought (eg, hypoglycemia, hyponatremia, hypocalcemia, hypomagnesemia). Many patients exhibit electrographic seizures that are undetectable by the clinician, even when the patients are not paralyzed. Therefore, clinicians should consider continuous EEG monitoring for the comatose postarrest patient. If the patient is under neuromuscular blockade, EEG monitoring will be the only means of detecting postischemic seizure.

XII. EVALUATION OF SUDDEN DEATH

Sudden death in infants, children, and adolescents may be due to underlying cardiac abnormalities, such as channelopathies, cardiomyopathies, and coronary artery abnormalities. Genetic mutations leading to channelopathies are relatively common among infants and children with out-of-hospital cardiac arrests. When a child or young adult has an unexplained sudden death, full medical and family histories should be obtained, and family members should be referred for evaluation of potential channelopathies. Patients should undergo a full echocardiography workup. In the event of death, an autopsy is recommended and, if possible, a genetic tissue evaluation should be performed.

XIII. ETHICAL ISSUES

A. Family Presence

The presence of the family at the bedside during resuscitation has become increasingly common. Those who are present during the resuscitation are able to witness the efforts utilized and, in the event of death, their presence has been shown to help with grieving and lessening of anxiety and depression. The option to be present should be offered to family members when possible. A member of the medical team should be assigned to support the family during the resuscitation and, if their presence is disruptive, the family respectfully should be asked to leave the room to facilitate the resuscitation efforts and the team dynamics.

B. Prognosis

Predicting outcome after resuscitation from cardiac arrest is a complex task. Much of the relevant data comes from adult patients who differ in arrest etiology and neurodevelopment. Early prognostication (within 24 hours of arrest) is limited by the fact that no single marker is 100% sensitive for poor outcome. Even fewer data are available to help predict a good outcome. Clinical neurologic findings of absent pupillary response and motor response predict outcome, but not until 3 days after the arrest. Neurophysiological diagnostic studies, such as EEG and somatosensory evoked potentials, may be helpful, but are not standardized in the pediatric population and not available at all centers. Computed tomography scans are not sensitive for early neurologic injury, but may be helpful in determining a cause of out-of-hospital arrest, including intracranial hemorrhage or intracranial hypertension. Magnetic resonance imaging with diffusion weighting provides valuable information about hypoxic/ischemic injury in the subacute and recovery phases (5-7 days after injury). Comparison to a child's prearrest neurologic function is difficult and adds another barrier to the assessment and prediction of postarrest neurologic status. The AHA 2015 guidelines addressed several intra-arrest and postarrest clinical variables for prognostic significance, however, no single variable was identified to be sufficiently reliable to predict outcomes. Therefore, the recommendation was to consider multiple factors that may predict outcomes during cardiac arrest and in the post-ROSC setting. Care providers should take hypothermia into account when making recommendations about withdrawal of care, and neurology consultation should be considered.

C. Termination of Resuscitative Efforts

No reliable predictor of outcome can determine when to terminate resuscitative efforts. Several factors point to the likelihood of survival after cardiac arrest, including the mechanism of the arrest (eg, traumatic, asphyxial, progression from circulatory shock), location (eg, in or out of the hospital), response (eg, witnessed or unwitnessed, with or without bystander CPR), underlying pathophysiology (eg, cardiomyopathy, congenital defect, drug toxicity, or metabolic derangement), and the potential reversibility of underlying diseases. Patients who have a witnessed arrest, receive bystander CPR, or receive advanced medical care a short time after collapse may have a better chance for a good outcome. When high-quality CPR is provided and rescue extracorporeal membrane oxygenation is available, even CPR lasting an hour may result in a good neurologic outcome. Therefore, it is important to consider the multiple variables in determining when to terminate efforts.

Key Points

Pediatric Cardiac Arrest

- Early identification of the decompensating patient is paramount to preventing cardiac arrest.
- Rapid initiation of chest compressions is critical to resumption of coronary blood flow and ROSC.
- Critical factors in the administration of high-quality CPR are push hard, push fast, minimize interruptions, allow full chest recoil, and avoid overventilation.
- If IV access is difficult to achieve, an intraosseous line should be placed immediately.
- In patients with VF, defibrillation should not be delayed for endotracheal intubation.
- Use of an AED should be considered in efforts to defibrillate children if a manual defibrillator is not available.
- Post-resuscitation assessment and management of temperature, glucose, oxygen saturation, $Paco_2$, and blood pressure may improve outcomes from cardiac arrest.

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DIAGNOSIS AND MANAGEMENT OF THE CHILD WITH ACUTE UPPER AND LOWER AIRWAY DISEASE

Objectives

- Detail the anatomic and developmental factors that make infants and children prone to disorders of the upper airway.
- Describe how to assess children with upper airway disorders.
- Summarize the diagnosis and treatment of children with upper airway disorders.
- Identify common disorders of the upper airway.
- Outline the pathophysiology of acute lower airway disease.
- Detect respiratory failure with or without accompanying respiratory distress.
- Initiate appropriate diagnostic and therapeutic interventions for the various causes of acute lower airway disease.

Case Study

A 9-month-old infant presents to the emergency department with sudden onset of noisy and difficult breathing. He appears anxious as he clings to his mother. You note a high-pitched noise upon inspiration, which is accompanied by subcostal, intercostal, and suprasternal retractions. He becomes upset and his work of breathing worsens whenever staff approach him for assessment.

Detection

– What conditions are in your differential diagnosis?

– What does the high-pitched noise signify?

Intervention

– What actions should be taken immediately?

Reassessment

– How do you know the interventions are effective?

– How should this patient be monitored?

Effective Communication

– Who needs to know about changes in the patient's clinical condition?

– Where is the best place to manage this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

I. UPPER AIRWAY DISEASE

Problems related to the upper airway occur frequently in infants and children. These problems range from benign and self-limiting, such as most cases of viral croup, to immediately life-threatening, as in cases of acute upper airway obstruction due to foreign body aspiration. Management strategies for upper airway-related disorders extend from calm reassurance to urgent life-saving intervention. Recognition of the etiologies of upper airway diseases in children, assessment of their severity, and approaches to treatment are important for treating critically ill hospitalized children.

II. ANATOMY OF THE PEDIATRIC AIRWAY

A. Airway

The pediatric airway undergoes changes between birth and approximately 8 years of age, when it becomes anatomically similar to the adult airway. Congenital abnormalities can affect function, in addition to disorders related to an innately small size. Pediatric patients are more susceptible to several illnesses, such as viral croup, than adults because the size of the airway. After children reach 8 years of age, their upper airway problems become similar to those found in adults.

Chapter 2 describes many of the developmental differences of the newborn, infant, pediatric, and adult airways and the impact of those differences on airway management. **Table 4-1** highlights some of those differences.

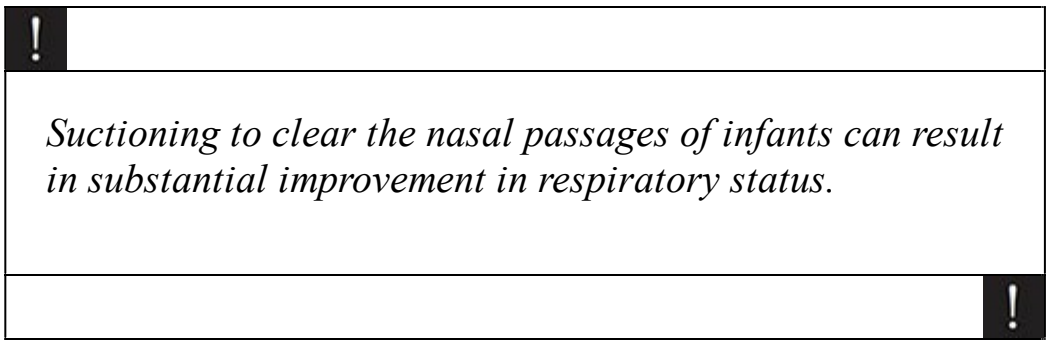
Table 4-1 ANATOMIC DIFFERENCE BETWEEN INFANT, PEDIATRIC, AND ADULT AIRWAYS

Characteristic	Infant	Pediatric (Age 1 Year)	Adult

Air entry	Obligate nasal breather	Mouth and nose	Mouth and nose
Tracheal diameter	4 mm	5-6 mm	18 mm
Laryngeal shape	Funnel	Funnel	Barrel
Narrowest point past cords	Cricoid	Cricoid	Laryngeal inlet
Epiglottis	Long, soft, omega-shaped	Long, soft, omega-shaped	Short, firm
Laryngeal inlet	C1	C3-C4	C5-C6
Proportion of tongue to jaw (jaw space)	Tongue relatively large in proportion to oral cavity	Proportion of tongue to jaw space changes continually up to approximately 8 years	Tongue easy to displace into jaw
Position of larynx on laryngoscopy	Anterior, cephalad	Transition between infant and adult position	Posterior, caudad
Trachea	Soft cartilage	Firmer but still collapsible cartilage	Firm, rigid tracheal cartilage

Between birth and the age of 3 to 6 months, infants are obligate nasal breathers, making them prone to distress from congenital and acquired nasal obstruction. Congenital causes of nasal obstruction include unilateral and bilateral choanal stenosis. Acquired causes include mucous plugging and edema from upper respiratory tract infections, such as those due to respiratory syncytial virus (RSV). Suctioning to clear the nasal passages of infants can substantially improve respiratory status. As a child ages, oral breathing becomes possible, but there continues to be a preference for the nose.

A small chin is developmentally appropriate at birth in that it facilitates both vaginal delivery and breast-feeding. Anatomically, a small chin is manifested by short mandibular rami and a short distance from chin to hyoid cartilage. This contributes to a smaller jaw space and is one reason that the tongue is relatively large for the size of the oral cavity around birth. A relatively large tongue-to-mouth ratio facilitates obligate nasal breathing, which, in turn, permits a child to feed without aspirating. The ratio of tongue size to oral cavity size decreases as a child grows. A large tongue in a relatively small mouth, though anatomically normal, makes infants with a decreased level of consciousness more prone to develop upper airway obstruction from the tongue falling back into the pharynx.



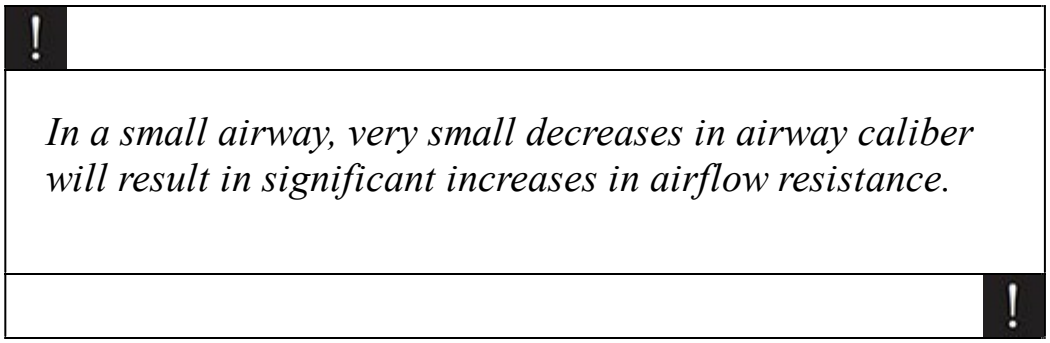
In the Pierre Robin sequence (micrognathia, relative macroglossia, cleft palate), the severe disproportion in size between the tongue and mouth may result in the tongue being pressed back into the pharynx, obstructing air movement. A small submental space in infants results in less space into which the tongue can be displaced during laryngoscopy and adds difficulty to intubation. Airway visualization by laryngoscopy is always more difficult in patients with micrognathia, macroglossia, or both.

As a child approaches 8 years of age, the narrowest point of the airway changes from the subglottic space to the laryngeal inlet—the relationship present in adult airways. The airway diameter of newborns, infants, and children increases with growth until the mid-teens, when it reaches adult caliber. The narrow airway diameter of infants and children results in increased resistance to airflow. Resistance to airflow is described mathematically by the Hagen-Poiseuille equation:

$$R = 8\eta l/r^4$$

where *R* is resistance, η is the viscosity of the gas, *l* is the length of tube that air flows through, and *r* is the radius of the airway. The equation illustrates that resistance goes up exponentially to the fourth power as airway diameter (twice the radius) decreases. Therefore, even though the absolute volume of gas flow is lower than in adults, resistance to airflow is greater in infants and children (**Chapter 2**).

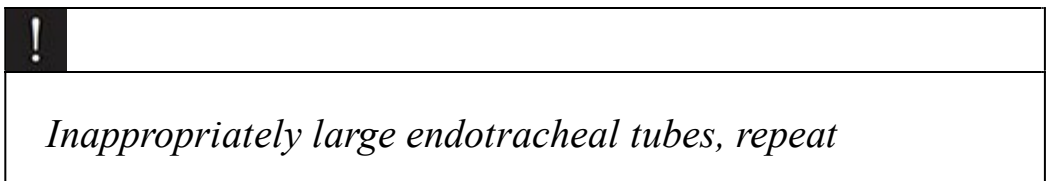
Figure 2-1 in **Chapter 2** illustrates the relative increase in resistance when 1 mm of circumferential airway edema occurs in an infant airway that is 4 mm in diameter, compared to the increase that occurs when the same amount of circumferential edema occurs in the 8-mm diameter airway of an older child or teen. Remembering that 1 mm of circumferential edema results in a 2-mm decrease in diameter and using Poiseuille's law, it is seen that the $1/r^4$ relationship of airway radius to resistance results in a 16-fold increase in airway resistance in the 4-mm airway compared to a 3-fold increase in resistance in the 8-mm airway.



In a small airway, very small decreases in airway caliber will result in significant increases in airflow resistance.

Until a child reaches approximately 8 years of age, the narrowest point of the airway after the vocal cords is in the subglottic space. Infant and pediatric larynges have a funnel shape rather than the barrel shape of an adult larynx. Thus, the adult airway is narrowest at the vocal cords, or laryngeal inlet, whereas the infant and pediatric airways continue to narrow below the vocal cords, reaching their narrowest portion at the level of the cricoid cartilage. With the narrowest portion of the upper airway located in the subglottic space, infants have a susceptibility to significant airflow compromise in response to a small volume of tissue edema. As the airway diameter increases with growth, the increases in resistance related to airway edema become less significant.

Although passing an endotracheal tube through the vocal cords always places a patient at risk for vocal cord injury, a narrow subglottic space makes infants and children susceptible to subglottic injury.



Inappropriately large endotracheal tubes, repeat

intubations, excessive movement, and thrashing while intubated all may contribute to subglottic injury and edema, which may result in scar tissue and lead to subglottic stenosis.

B. Epiglottis

The epiglottis of infants and young children differs in shape and consistency from an adult epiglottis. It is longer and softer than in the adult and assumes an omega-like shape (Ω). The adult epiglottis, in contrast, is relatively shorter, firmer, and more square than in children. The characteristics of the infant larynx necessitate that the epiglottis be actively displaced from the glottic opening during laryngoscopy for tracheal intubation as opposed to manipulating the vallecula, as is done in adults. The shape of an infant's epiglottis, when exaggerated, contributes to laryngomalacia. In this condition, an infant shows signs of inspiratory upper airway obstruction with attendant stridor because of the collapse of the epiglottis and laryngeal components over the laryngeal inlet on inspiration.

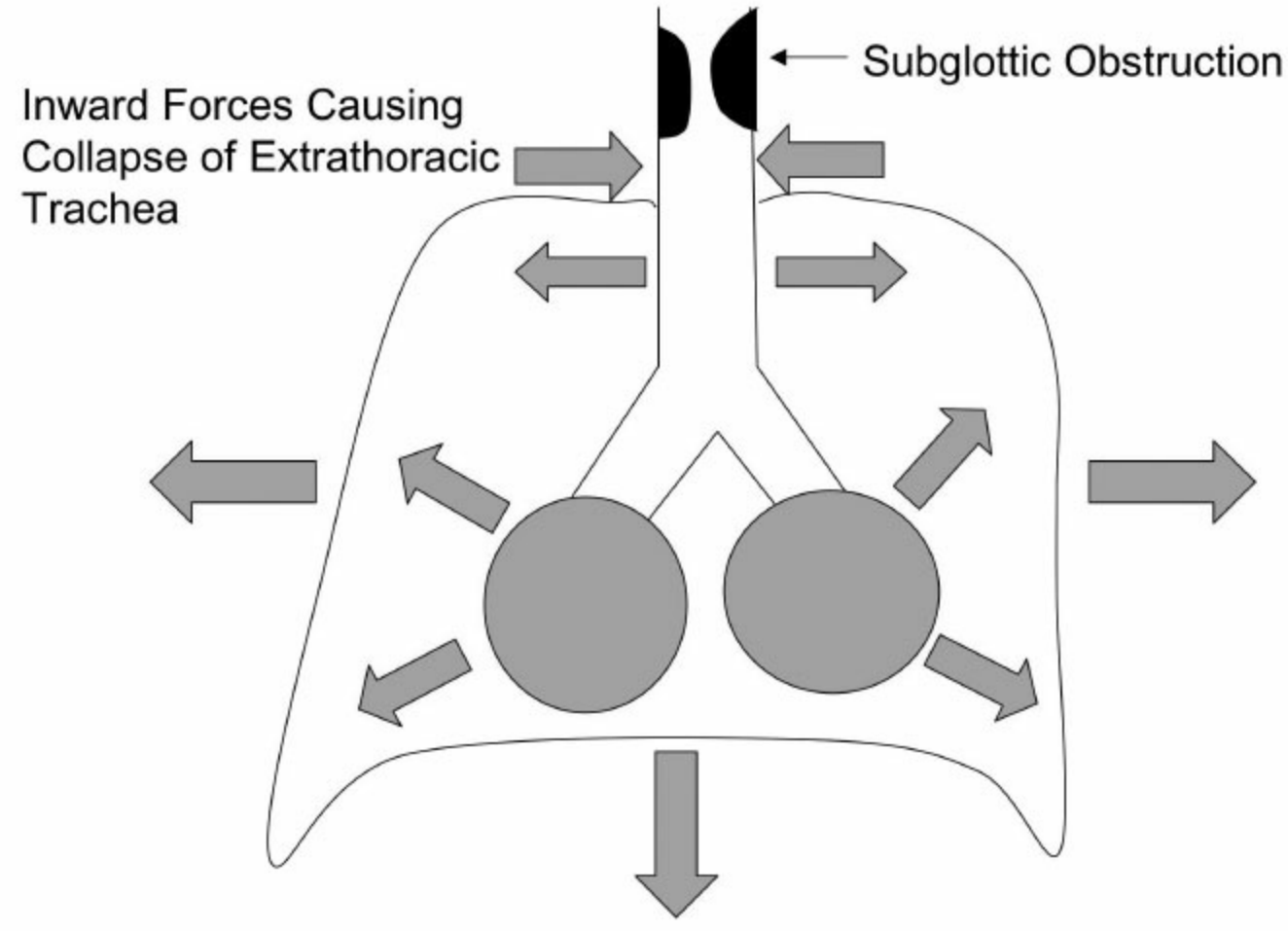
C. Larynx

The larynx of infants and young children is anterior and cephalad in position compared with the larynx of an adult. The impact of this relationship on airway visualization during intubation is discussed in **Chapter 2**.

D. Trachea and Lower Airways

Tracheal cartilage is soft and collapsible in infancy, becoming more rigid with growth. Thus, the walls of the extrathoracic trachea can collapse inward when an infant creates significant negative inspiratory force by inhaling against an obstructed upper airway (**Figure 4-1**).

Figure 4-1. Extrathoracic Tracheal Collapse with Forceful Inspiration Against a Subglottic Obstruction



The extrathoracic and intrathoracic portions of the trachea react differently to the forces generated by breathing. The portion of the trachea from the subglottic space to the thoracic inlet is extrathoracic. The trachea from the thoracic inlet to the carina is intrathoracic and sits within the chest, surrounded by lung and pleura, making it subject to the forces generated within the chest during inspiration and expiration. The intrathoracic trachea, as well as the bronchi and all the distal airways, are connected to the surrounding lung parenchyma, which in turn is covered by the visceral pleura. The visceral pleura of the lungs are apposed to the parietal pleura lining the inside of the rib cage by a mechanism known as *viscoelastic coupling*. Natural outward elastic recoil of the rib cage, along with the constant tone of the diaphragm, combine to transmit a force to the intrapleural space that results in negative intrapleural pressure at all times. The outward tension of the rib cage, the downward tone of the diaphragm, and a negative intrapleural pressure—all occurring at rest—combine to keep the lungs expanded and, because the airways are connected to the lung parenchyma, the airways are held open as well.

The pleural pressure, which is difficult to measure, is analogous to intrathoracic pressure. During quiet inspiration, the pleural (intrathoracic) pressure becomes more negative when the diaphragm contracts. The negative pleural pressure creates a pressure gradient from the alveoli (where it is most negative) to the mouth (where it is approximately zero). The pressure gradient drives airflow into the lungs. When the diaphragm contracts (as well as the accessory muscles of respiration, when the child is in distress), the negative intrathoracic pressure is transmitted to the outside of the trachea, serving as a distending force to keep the intrathoracic airways open. Increased airway resistance during inspiration due to partial or complete airway obstruction causes the diaphragm to contract more forcefully and the accessory muscles of inspiration (external intercostals, scalenes, and

sternocleidomastoids) to engage. The negative intrapleural and thus intrathoracic pressures will increase in an effort to move air past the obstruction. Due to the connection of the airways to the lung parenchyma (known as *interdependence*) and the lungs to the pleura, the more negative intrathoracic and intrapleural pressure will tend to keep the intrathoracic airways open; at the same time, the extrathoracic trachea—without the surrounding lung parenchyma to pull it open—will tend to close.

Intrathoracic and intrapleural pressures become very positive during forced expiration, as occurs in obstructive diseases such as asthma. When the intrapleural pressure becomes positive, the airway pressure gradient becomes very positive in the alveoli and decreases as it moves toward the mouth, where it is zero. A gradient is necessary for air movement to occur. As the pressure gradient decreases toward the mouth, there may come a point, known as *the equal pressure point*, where the pressure in the airways becomes equal to the intrapleural pressure. At any point closer to the mouth than the equal pressure point, the sum of forces favors closure of the airways.

Thus, the intrathoracic airways, including the intrathoracic trachea, will remain open when inspiratory effort is increased but are prone to closure when expiratory forces are increased. The extrathoracic trachea behaves in a manner opposite to that of the intrathoracic trachea. In the extrathoracic trachea, the outer trachea is not tethered to the lung and pleura to create a distending force to counterbalance the negative intratracheal pressure that occurs during inspiration. During quiet breathing, and when the tracheal cartilage is formed normally, no significant tracheal collapse occurs during inspiration; however, when a patient breathes against a partial upper airway obstruction, as occurs in laryngomalacia or viral croup, the tracheal walls can partially collapse, further increasing airway resistance. Collapse of the extrathoracic trachea during inspiration and of the intrathoracic trachea during expiration is much worse in the presence of poor cartilaginous ring development, as is seen in tracheomalacia.

!

Changes in Pediatric Airway With Partial Obstruction
Airflow restriction is related to the location of the obstruction.
Extrathoracic obstruction → Inspiratory collapse → Stridor
Intrathoracic obstruction → Expiratory airway collapse → Wheezing on expiration
In severe cases (eg, laryngotracheobronchitis), both may be present.

!

The extrathoracic trachea is not subject to collapse during forced exhalation. The differences between the intrathoracic and extrathoracic airways provide the basis for understanding why problems of the nose, pharynx, larynx, and subglottic trachea have their greatest impact on inspiration, whereas problems of the lower (intrathoracic) airways tend to have greater impact upon exhalation. A tendency for the subglottic, but extrathoracic, trachea to narrow or close in the presence of very

negative intrapleural pressure also explains why patients with upper airway obstruction do worse when they are agitated (**Figure 4-1**).

III. PEDIATRIC AIRWAY DISORDERS

Table 4-2 lists some of the disorders that may present as partial or complete obstruction of the upper airway in children. General approaches to these conditions are discussed in greater detail in a later section.

Table 4-2 CAUSES OF PEDIATRIC AIRWAY DISORDERS

Anatomic Disorders	Infectious Disorders
• Altered level of consciousness (airway muscle laxity) • Tonsillar hypertrophy • Subglottic stenosis (acquired or congenital) • Macroglossia • Vocal cord paralysis • Micrognathia • Choanal stenosis • Pierre Robin sequence • Cartilaginous deficiency: laryngomalacia, tracheomalacia, bronchomalacia	• Laryngotracheobronchitis (croup) • Epiglottitis (supraglottitis) • Bacterial tracheitis • Retropharyngeal (parapharyngeal) abscess • Peritonsillar abscess • Infectious mononucleosis
External or Internal Compression Disorders	**Miscellaneous Disorders**
• Tumor • Hemangioma • Hematoma • Cyst • Papilloma • Vascular rings and slings	• Airway trauma • Post-extubation airway obstruction • Angioedema • Spasmodic croup • Foreign body aspiration • Airway trauma • Thermal and chemical burns

A. Anatomic Disorders

1. Altered Level of Consciousness

An altered level of consciousness is frequently responsible for upper airway obstruction in patients of all ages. For developmental reasons, young patients are more prone to this. For example, the decreased level of consciousness that accompanies the postictal state after a seizure, especially when barbiturates or benzodiazepines have been administered, can lead to upper airway obstruction. Many decreases in oxygen saturation that occur during procedural sedation are related to obstruction. A child who has altered consciousness after an automobile or bicycle collision is likely to have a partially obstructed airway and may benefit significantly from maneuvers that open and maintain airway patency while protecting the neck's spinal alignment (**Chapters 2** and **9**).

2. Tonsillar-Adenoidal Hypertrophy

Tonsillar-adenoidal hypertrophy is common and reaches peak occurrence in children aged 4 to 8 years. Alone and combined with obesity, it is responsible for obstructive sleep apnea and acute upper airway obstruction. Though apnea is mostly a chronic problem, there are situations in which patients develop acute obstruction. Positive pressure ventilation by continuous positive airway pressure (CPAP) or other modalities of noninvasive ventilation (NIV) are often helpful. In an acute emergency, a nasopharyngeal airway may be employed. Tonsillectomy and adenoidectomy, along with weight loss, are the definitive treatments. Patients may continue to require positive airway pressure for some time after tonsillectomy and adenoidectomy. In acute situations, corticosteroids may be helpful in reducing tonsillar tissue size, especially when the cause is infectious mononucleosis.

3. Subglottic Stenosis

Subglottic stenosis (SGS) can be congenital or acquired. Acquired SGS is frequently seen in infants with a history of prematurity and intubation or in any patient with a history of repeated, prolonged, or traumatic intubation. Sometimes an acute intercurrent viral respiratory infection, especially RSV, can cause SGS to change rapidly from a chronic compensated problem to uncompensated respiratory distress. Racemic epinephrine, dexamethasone, and heliox are all useful interventions for ameliorating a severe event, but a patient with severe SGS ultimately requires airway endoscopy for a definitive diagnosis and, potentially, laryngotracheal reconstruction.

4. Macroglossia and Micrognathia

Macroglossia (large tongue) and micrognathia (small chin and jaw) are components of several congenital craniofacial anomalies. Their roles in upper airway obstruction were presented previously. Severe macroglossia and micrognathia can result in critical airway issues in the perinatal period. These issues become prominent when a patient requires procedural sedation or anesthesia with tracheal intubation.

5. Vocal Cord Paralysis

Vocal cord paralysis can be either unilateral or bilateral. Unilateral vocal cord paralysis is manifested by weak cry and occasional stridor, and is usually well tolerated. The etiology is often idiopathic, but the condition is occasionally associated with injury to the recurrent laryngeal nerve during thoracic surgery or may result from direct injury from intubation. Bilateral vocal cord paralysis manifests as severe stridor and severe respiratory distress, though often with a normal cry. Bilateral vocal cord paralysis is often associated with central nervous system injury or disease. Children with bilateral vocal cord paralysis often require urgent airway management.

6. Choanal Stenosis and Choanal Atresia

Choanal stenosis and choanal atresia may be unilateral or bilateral. Given that infants are obligate nasal breathers, those with choanal stenosis develop some degree of distress and poor feeding. Bilateral choanal stenosis almost always produces symptoms during the neonatal period. The diagnosis is confirmed by attempting to pass a soft catheter through the nose. Evaluation of the nose

and nasopharynx by computed tomographic scanning also is helpful in confirming the diagnosis and in determining the extent of obstruction. Associated anomalies occur in 20% to 50% of infants with choanal atresia. The syndrome of anomalies, known as the *CHARGE association*, includes coloboma and other ophthalmologic anomalies, heart disease, choanal atresia, growth retardation, genital hypoplasia, and ear anomalies. Choanal stenosis and choanal atresia are treated surgically.

Unilateral choanal stenosis may make it difficult or impossible to pass a nasogastric tube through the stenotic nares. If the tube is instead passed through the unobstructed nostril, an infant will become distressed due to occlusion of the one patent naris.

7. Pierre Robin Sequence

The Pierre Robin sequence is an example of a congenital syndrome in which upper airway obstruction can be life-threatening. The primary defect is poor mandibular growth in utero that results in micrognathia, cleft palate, and glossoptosis (posterior displacement or retraction of the tongue). The oral cavity is too small for the tongue, which is usually a normal size. As a result of the mismatch between tongue and jaw sizes, affected infants are prone to airway obstruction from posterior movement of the tongue against the retropharyngeal wall. Approaches to management include prone positioning to allow the tongue to move forward and away from the pharynx. The placement of a nasopharyngeal (NP) tube made from an appropriately sized endotracheal tube can be lifesaving. The stiffness of the endotracheal tube lends itself to better airway patency. The general technique for using NP tubes is presented in **Chapter 2**, but the approach in **Figure 4-2** focuses on differences specific to the Pierre Robin sequence.

Figure 4-2. Placement of Nasopharyngeal Tube for Airway Obstruction Due to Pierre Robin Sequence

1. Use an uncuffed tracheal tube that fits in a nostril.
2. Match the length of the tube to the approximate distance between the naris and the tragus.
3. Lubricate the tube with surgical lubricant.

4. Insert and advance the tube while listening for air movement.
5. Secure the tube carefully with tape and adhesive.
6. Suction frequently to maintain patency.
7. If air movement through the tube cannot be heard, reposition the tube until airflow is audible.
8. Use of in-line capnography can be invaluable in assessing airway patency.

When air movement through the NP tube is heard and felt, the tube is in the appropriate position. This will lessen the patient's distress. The tube should be secured at this position with tape and adhesive in the same way as an endotracheal tube. Though it does not pass through the vocal cords and into the trachea, the NP tube is nonetheless a lifeline through which the patient can move air. The tube should be suctioned as necessary to maintain patency. Should the patient develop distress, suctioning and, if necessary, repositioning of the tube are indicated until air movement is again detected. The tube has a dual purpose: it serves as a conduit for air movement but also pushes the tongue slightly forward off the retropharyngeal wall, thus relieving that level of obstruction.

An additional lifesaving maneuver for severe cases of obstruction in the Pierre Robin sequence is to pull the tongue forward manually using a towel clamp or similar instrument. Passing a needle with size 0 silk through the tip of the tongue and pulling it forward and out of the mouth may serve a similar purpose. Definitive management for severe cases is surgical, occasionally with a need for tracheostomy.

B. External or Internal Compression Disorders

Case Study

A previously healthy 10-year-old girl is evaluated in the emergency department for gradual new-onset wheezing, increased work of breathing, and increased dyspnea in a supine position. Her mother also reports a temperature of 39°C (102.2°F), weight loss, and night sweats. The child prefers the sitting position and looks anxious. On exam, her vital signs are as follows: heart rate, 120 beats/min; respiratory rate, 25 breaths/min; blood pressure, 130/75 mm Hg; and pulse oximetry 91% on room air. Inspiratory and expiratory wheezes are found on auscultation with decreased breath sounds in the right upper chest. Despite your recommendation, the patient is placed in a supine position. She becomes more anxious, her oxygen saturations drops, and the engorgement of her neck veins increases.

Detection

– What is happening to this patient?

– What is the differential diagnosis?

– Why did she worsen when placed supine?

– What tests could be useful to evaluate this patient?

Intervention

– What interventions should be made immediately?

Reassessment

– How will you know if the interventions are working?

– How should the patient be monitored?

– Are any laboratory studies needed?

Effective Communication

– Who needs to know about this patient immediately?

– What information needs to be communicated at each stage of movement in the hospital?

– Where is the best place to care for this patient?

Teamwork

– How is the work to be distributed?

1. Diagnosis of Neoplastic Airway Obstruction

External or internal compression of the airway can result in upper airway obstruction. Tumors, hemangiomas, hematomas, cysts, papilloma, and vascular rings and slings can all present with signs and symptoms of upper airway obstruction. Lymphoid (Hodgkin and non-Hodgkin lymphoma) and leukemic (acute lymphocytic leukemia) malignancies in children may present with an anterior or middle mediastinal mass. Tumor masses in this location produce superior vena cava obstruction and compression of other mediastinal structures, such as the trachea, main stem bronchus, pulmonary vessels, and aorta. Progressive venous congestion and airway compression will lead to the usual symptoms found in superior vena cava obstruction: facial engorgement, headache, plethora, cyanotic facies, cough, dyspnea, orthopnea, hoarseness, and dysphagia.

Sedation and airway instrumentation of children with neoplastic airway obstruction should be performed by experienced anesthesiologists or intensivists. The use of sedatives and muscle relaxants can precipitate a total airway collapse.

Posterior, anterior, and lateral chest radiographs will show a widening of the mediastinum with tracheal deviation or compression.

2. Management of Neoplastic Airway Obstruction

NIV can temporarily reduce the degree of airway obstruction and improve airflow. When either is started, it is helpful to auscultate for breath sounds over the lung fields while titrating pressures to find one that opens the airway. In the presence of an anterior mediastinal mass, positioning the patient upright, prone, or even on the side can take pressure off the trachea and relieve obstruction. An experienced anesthesiologist and an ear, nose, and throat surgeon should be available in the event that endotracheal intubation is necessary. The patient's airway is best secured with the patient breathing spontaneously, as catastrophic airway closure can occur with deep sedation or paralysis.

C. Infectious Causes of Upper Airway Obstruction

Infectious causes of upper airway obstruction are common in patients of all ages (**Table 4-3**). The past 20 years have seen a reduction in the incidence of epiglottitis due to the addition of the *Haemophilus influenzae* type b (Hib) vaccine in the infant immunization schedule in the United States and developed countries. Prior to vaccine use in the United States, the incidence of epiglottitis was 5 cases per 100,000 children who were 5 years and younger; this incidence decreased to 0.6 to 0.8 cases per 100,000 children after the addition of Hib vaccination. *H influenzae* is a fulminant and life-threatening infection requiring knowledge of its diagnosis and management. Moreover, the evaluation and care of the patient with epiglottitis often serves as a paradigm for the approach to all problems that can result in severe upper airway obstruction.

Table 4-3 COMMON INFECTIOUS CAUSES OF UPPER AIRWAY OBSTRUCTION

Characteristic	Croup	Epiglottitis	Bacterial Tracheitis	Retropharyngeal Abscess
Onset	Gradual, viral prodrome, 1-7 days	Rapid onset, 6-12 hours	Viral prodrome followed by rapid deterioration	Viral prodrome followed by rapid deterioration
Typical age at onset	6 months-4 years	2-8 years	6 months-8 years	<5 years
Seasonal occurrence	Late fall-winter	Throughout the year	Fall-winter	Throughout the year
Causative agents	Parainfluenza, respiratory syncytial virus, influenza A	*Haemophilus influenzae* type B (classically), *Streptococcus pneumoniae*, GABHS	*Staphylococcus aureus* (classically), GABHS, *Streptococcus pneumoniae*	Anaerobic bacteria, GABHS, *Staphylococcus aureus*
Pathology	Subglottic edema	Inflammatory edema of supraglottis	Thick, mucopurulent, membranous tracheal secretions	Abscess formation in deep cervical fascia
Fever	Low grade	High	High	High
Cough	Barking or seal-like	None	Usually absent	Usually absent

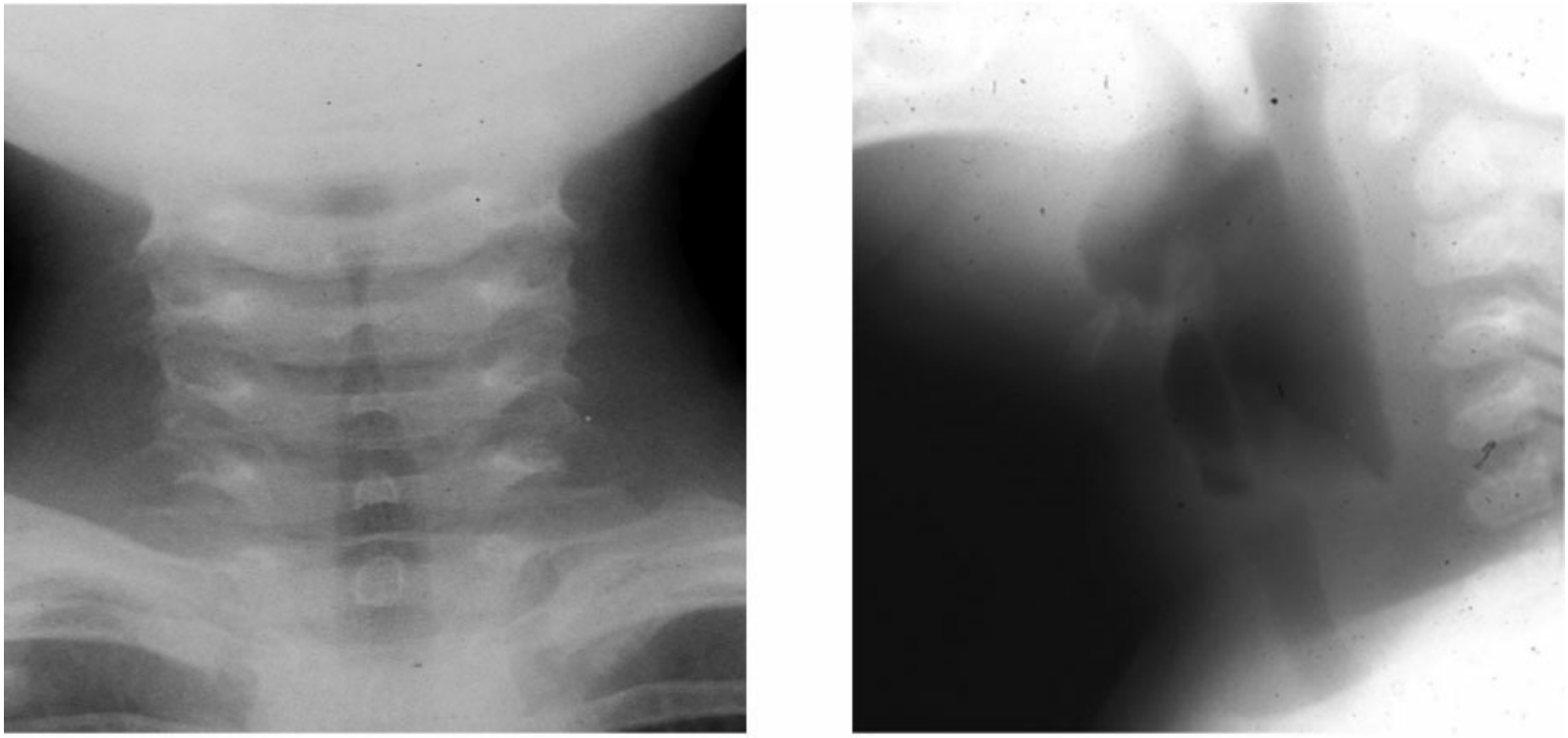
Sore throat	None	Severe	None	Severe
Drooling	None	Frequent	None	Frequent
Posture	Any	Sitting forward, mouth open, neck extended (tripod position)	Any	Sitting forward, mouth open, neck extended (tripod position)
Voice	Normal or hoarse	Muffled	Normal or hoarse	Muffled
Appearance	Nontoxic	Toxic	Toxic	Toxic

GABHS, group A b-hemolytic streptococci

1. Viral Laryngotracheobronchitis (Croup)

Viral laryngotracheobronchitis (croup) is the most common infectious cause of upper airway obstruction in children, primarily affecting children aged 6 months to 4 years, with a peak incidence between 1 and 2 years of age in North America. Cases of croup can be seen throughout the year, with a peak incidence during fall and early winter. It is most commonly caused by parainfluenza virus type 1, though types 2 and 3, *H influenzae* types a and b, RSV, adenovirus, and *Mycoplasma pneumoniae* may all cause croup. Most children are managed as outpatients, but some do require hospitalization, and about 1% of hospitalized children require tracheal intubation and mechanical ventilation. Children with croup usually present after several days of prodromal viral-like symptoms (cough, coryza, rhinorrhea, low-grade fever) with progressively worsening hoarseness, eventually developing a classic seal-like, barking cough and stridor. Stridor is classically inspiratory but may be biphasic, indicating airway edema that extends beyond the subglottic space into the intrathoracic trachea and bronchi. Radiographs of the airway (**Figure 4-3**) may demonstrate a classic steeple sign (narrowing of the subglottic trachea coming to a point at the level of the vocal cords). Lateral neck films in croup may show dilatation of the airway above the level of the vocal cords.

Figure 4-3. Laryngotracheobronchitis (Croup)



A, Anteroposterior radiograph demonstrating narrowing of the airway in a steeple-like manner that is characteristic of viral laryngotracheobronchitis. **B**, A lateral neck radiograph of a patient with viral croup shows a sharp epiglottis, characteristic dilation of the airspace above the level of the vocal cords, and narrowing of the subglottic air column.Courtesy of Terry L. Levin, MD, Children's Hospital at Montefiore Medical Center, New York, New York, USA.

Croup is generally self-limiting and requires only supportive care with cool mist and IV fluids if a patient is unable to drink. Nebulized racemic epinephrine rapidly reduces airway edema and improves symptoms. The reduction in edema is limited by the lifetime of the drug, and symptoms usually return within 2 hours. Corticosteroids have been shown to bring about significant improvement and are prescribed routinely. Heliox may be helpful. Nasal CPAP and NIV have been used with some success and may hold off tracheal intubation. In the event that intubation is required, an uncuffed, smaller than normal tube should be used as these patients are increased risk for development of subglottic stenosis. The patient should remain intubated until an air leak develops around the tube. The treatment of croup is summarized in **Table 4-4**.

Table 4-4	TREATMENT OF CROUP

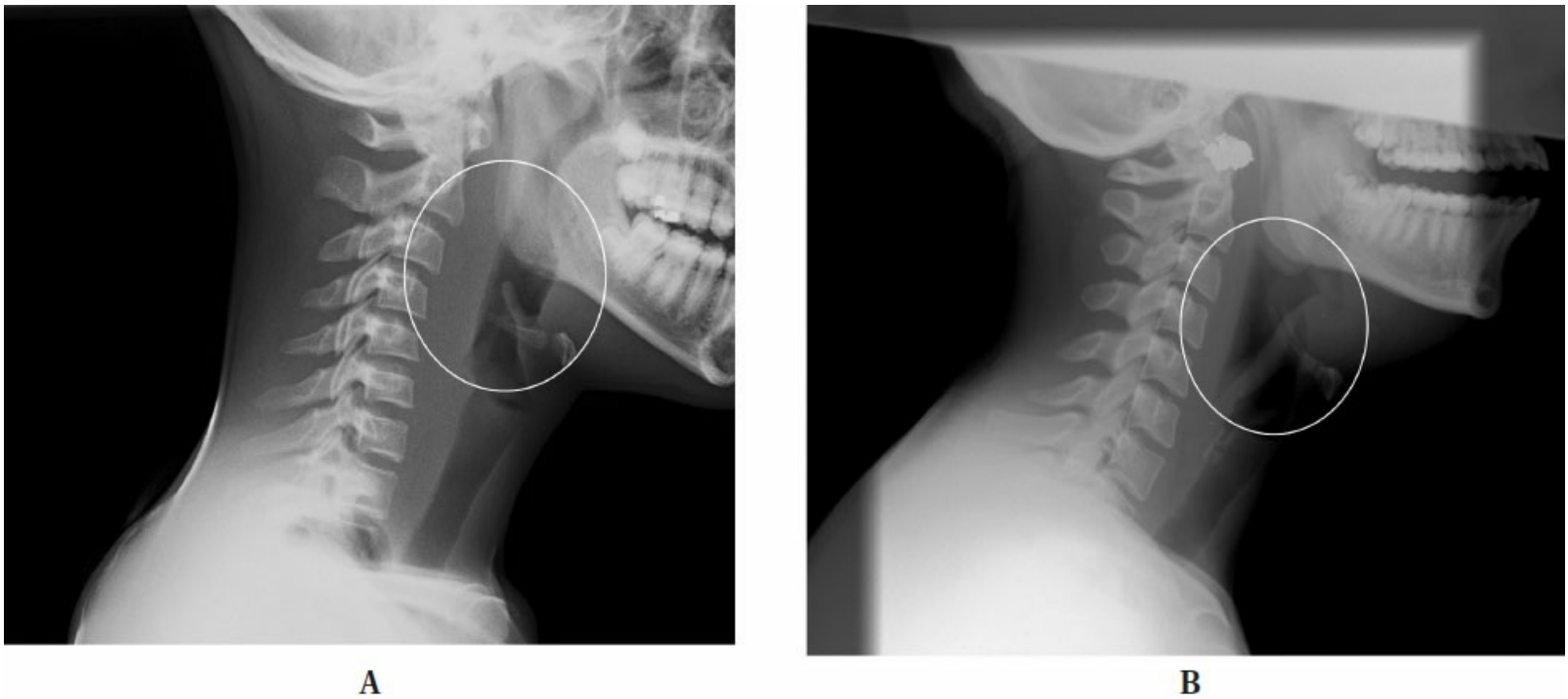
- Minimize situations that may cause distress in the child, such as separation from parents and unnecessary examination and procedures.
- Provide blow-by oxygen if desaturation or cyanosis is present.
- Give oral, intramuscular, or IV dexamethasone, 0.6 mg/kg.
- Administer nebulized racemic epinephrine (adrenaline), 2.25%, 0.5 mL in 2.5 mL saline.
- If the response is good, observe the patient for 2 hours and reevaluate for return of symptoms.
- If the response is poor or if stridor rapidly recurs, admit the patient to the ICU for observation.
- Consider heliox in a 60:40 or 70:30 mixture.
- Consider nasal continuous positive airway pressure or bilevel positive airway pressure.
- If intubation is required, select a tube that is smaller than normal for the patient's age (uncuffed preferred).

2. Epiglottitis (Supraglottitis, Panglottitis)

Epiglottitis is a true emergency. Though it is rare due to widespread use of the Hib vaccine, having knowledge of this life-threatening infection is still very important. The name implies involvement of only the epiglottis, but because all of the supraglottic structures can be involved, the term *supraglottitis* or *panglottitis* is more correct. Historically, epiglottitis was most commonly caused by *H influenzae* type b and was associated with bacteremia and severe toxicity. However, in the era of widespread Hib vaccination, the incidence of pediatric invasive *H influenzae* type b infections has dropped from 60 cases per 100,000 children to 0.21 per 100,000. *Streptococcus pneumoniae*, group A β-hemolytic streptococci (GABHS), and *Staphylococcus aureus* now are more common causes. The timing of the disease has also shifted, now affecting mostly older children and adults.

Children with supraglottitis usually present with rapidly progressive signs and symptoms, including high fever, irritability, drooling, dysphagia, dyspnea, and dysphonia. They appear toxic and prefer to rest in the tripod position. Stridor occurs late in the course and is usually softer than the loud stridor that occurs with croup. A lateral neck radiograph can be obtained if the suspicion of epiglottitis is low but cannot be totally ruled out; however, it should be deferred if it increases the child's level of anxiety or will delay definitive diagnosis and management. If epiglottitis is present, on lateral view, the epiglottis appears rounded, large, and thumb-shaped in comparison to a normal epiglottis, which is sharp (**Figure 4-4**).

Figure 4-4. Radiographs of Healthy Patient and Patient with Epiglottitis



A, Healthy patient shows a sharply defined epiglottis (circled). **B**, Patient with epiglottitis demonstrates an edematous, less clearly defined, thumb-like epiglottis (circled), as well as edema of the aryepiglottic folds and other supraglottic tissues consistent with supraglottitis.
Courtesy of Terry L. Levin, MD, Children's Hospital at Montefiore Medical Center, New York, New York, USA.

Even if the likelihood of epiglottitis is low, a healthcare provider trained in airway support and bag-mask ventilation, and who has the necessary emergency airway equipment, must accompany a child transferred to the radiology department for lateral neck radiographs. If epiglottitis is suspected based on history and physical exam, the patient should instead be transferred to surgery, accompanied by a

provider skilled in airway management and all necessary airway support equipment. Aside from anesthesiology, a surgeon capable of emergent surgical airway intervention should be alerted and immediately available if the case progresses to a failed airway. The management of suspected epiglottitis is summarized in **Table 4-5**.

Table 4-5	EPIGLOTTITIS

- Low suspicion of epiglottitis → send patient to radiology department for lateral neck radiographs
- High suspicion of epiglottitis → send patient to surgery for direct examination under anesthesia and intubation
- Whether suspicion is low or high, the patient should be:
  – Accompanied by a physician skilled in airway management
  – Kept in a comfortable position
  – Transported with a bag-mask ventilator, a mask of appropriate size and, if possible, a positive end-expiratory pressure valve

All noxious interventions, such as IV insertion, blood sampling, and examination of the pharynx, should be deferred until the child is sedated in the operating room and the airway has been secured by tracheal intubation. The child should be permitted to remain in a position of comfort during transport and mask induction of anesthesia. Ensure the child is spontaneously breathing under anesthesia before visualizing the larynx via direct laryngoscopy. Tracheal intubation is performed if epiglottitis is confirmed. The endotracheal tube should be secured well before transfer to the ICU. An IV should be inserted and blood obtained, and samples from the epiglottis, tracheal secretions, and blood should be sent for culture. Broad-spectrum antibiotics effective against β-lactamase-producing organisms should be administered. Antibiotic choices include second- and third-generation cephalosporins, such as cefuroxime or ceftriaxone or, alternatively, ampicillin-sulbactam. Improvement is usually rapid once antibiotics are started. The patient can be extubated when an audible air leak develops around the tracheal tube.

3. Bacterial Tracheitis

Bacterial tracheitis, a relatively rare but potentially life-threatening cause of upper airway obstruction in children, can result in pediatric airway emergencies requiring admission to the ICU. In bacterial tracheitis, thick mucopurulent membranous secretions obstruct the airway and cannot be cleared by cough, so tracheal intubation is usually required. Children typically present with a viral prodrome of several days' duration that has developed into a croup-like illness with low-grade fever, cough, and stridor. This illness then worsens rapidly, and the patient develops high fever, signs of increasing respiratory distress, an inability to clear secretions, and a toxic appearance. Radiographic imaging may show a croup-associated steeple sign. Additionally, the tracheal air column may display an edematous or ragged appearance. The similarity of the prodromes of croup and bacterial tracheitis has led to an impression that bacterial tracheitis is a bacterial superinfection of viral croup. Data from the United States, Australia, and Great Britain confirm that bacterial tracheitis affects children aged 6 months to 8 years, with a peak incidence during the fall and winter, corresponding to the viral respiratory season.

Children who present to an emergency department with bacterial tracheitis may be indistinguishable from those with epiglottitis and therefore should be managed according to a suspected epiglottitis

protocol. Direct inspection of the airway usually reveals subglottic edema with ulcerations, erythema, and pseudomembrane formation in the trachea. Historically, *S aureus* is the most common causative agent, but *S pneumoniae*, GABHS, and viruses have also been identified. On occasion, a patient who has been in an ICU with croup-like symptoms will acutely worsen and require tracheal intubation, resulting in copious thick secretions that come up through the tracheal tube and lend support to a diagnosis of bacterial tracheitis.

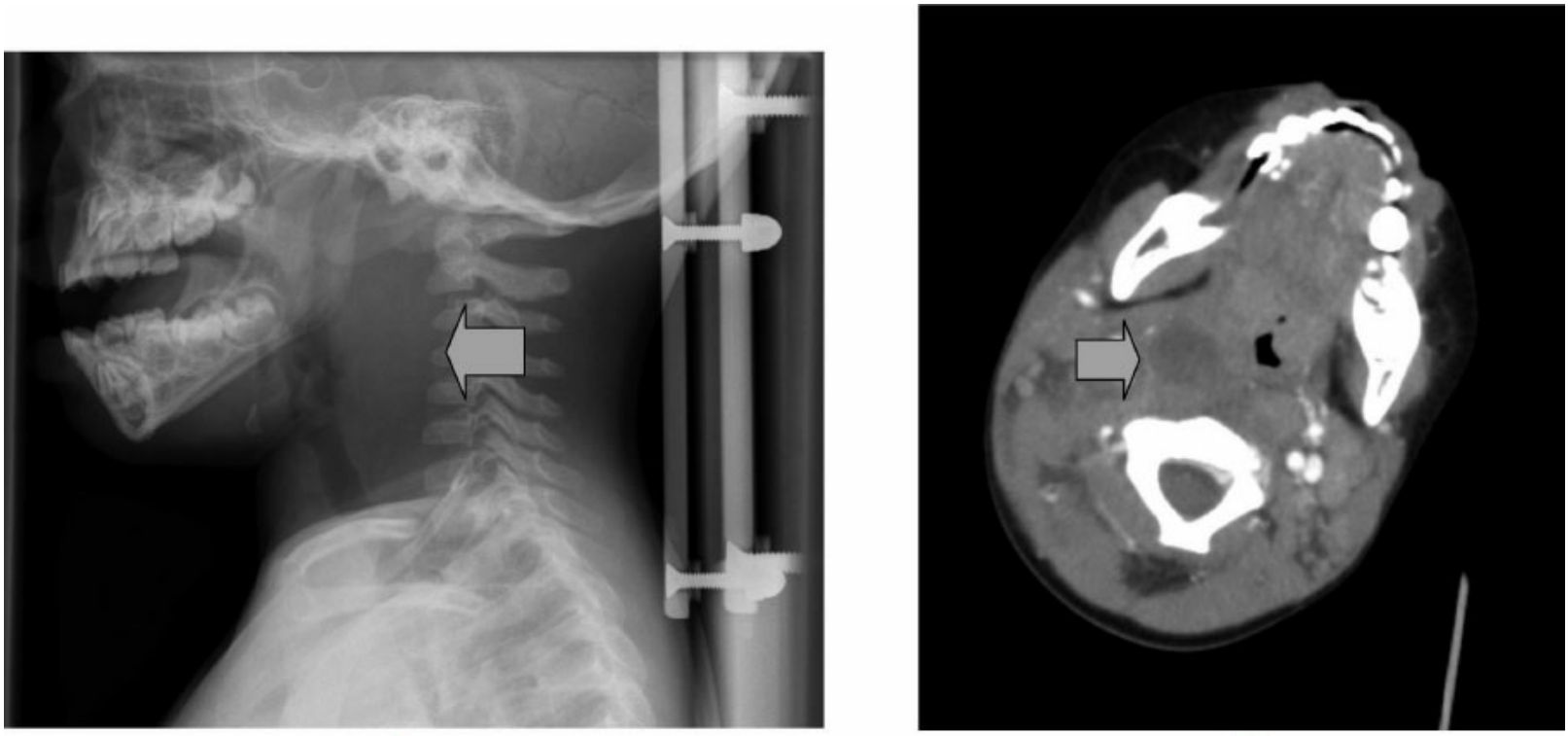
Pulmonary and tracheal toilet, tracheal intubation, and broad-spectrum antibiotics directed against *Staphylococcus* and *Streptococcus* species are the basics of treatment. A second-generation cephalosporin, such as cefuroxime, is a reasonable choice of antibiotic. Extubation may be attempted following clinical improvement and development of an air leak around the tracheal tube. This may take 3 to 5 days.

4. Retropharyngeal Abscess (Parapharyngeal Abscess)

Retropharyngeal abscess is an infection of the soft tissues of the posterior pharyngeal wall. The retropharyngeal space consists of a loose network of connective tissues and lymph nodes that drain the nasopharynx, paranasal sinuses, middle ear, teeth, and facial bones. Infection and abscess formation in this area usually result from lymphatic spread of infection or direct spread from the nasopharynx, paranasal sinuses, or middle ear. These lymph nodes atrophy during early childhood, thus decreasing the risk of disease in older children and adolescents. For this reason, direct trauma to the posterior pharynx from a pencil or stick in the mouth and ingestion of foreign bodies account for the majority of cases in older children and adolescents. Most cases occur in children younger than 5 years, thereby overlapping the most common ages for epiglottitis and bacterial tracheitis.

Children with retropharyngeal abscess have a nonspecific collection of symptoms that eventually coalesce as high fever, sore throat, and stiff neck. Symptoms are similar to those of epiglottitis, and physical exam may reveal a bulging unilateral neck mass. Neck stiffness may mimic that seen in meningitis. To some extent, this may be differentiated by having the patient move the neck in extension and flexion. A meningitic patient that will avoid flexion, but patients with a significant abscess will avoid neck extension. Presentation with torticollis is also common. The diagnosis may be confirmed by the presence of an increased prevertebral soft tissue space on lateral neck radiograph (**Figure 4-5**). The radiograph may also show the presence of gas or air fluid levels in the retropharyngeal space, as well as loss of the normal cervical lordosis. Retropharyngeal abscess is usually a polymicrobial infection. The cultures often yield anaerobic organisms, such as *Prevotella, Porphyromonas, Fusobacterium*, and *Peptostreptococcus*. Additionally, *S aureus*, GABHS, and *H influenzae* may be identified.

Figure 4-5. Retropharyngeal Abscess



A, Radiograph shows a widened distance between the air column and the anterior aspect of the spinal column (prevertebral space) [arrow]. **B**, Computed tomography image reveals a retropharyngeal abscess (arrow).
Courtesy of Terry L. Levin, MD, Children's Hospital at Montefiore Medical Center, New York, New York, USA.

Management of retropharyngeal abscess includes close observation and broad-spectrum antibiotics that have anaerobic coverage. Abscess drainage is recommended for cases refractory to antibiotics. Complications are rare with early recognition and appropriate antibiotic treatment, but serious complications include rupture of the abscess into the posterior pharynx (resulting in aspiration of infectious material), lateral spread of infection, and dissection into the posterior mediastinum through fascial planes. Deaths have been reported.

5. Peritonsillar Abscess (Quinsy)

Peritonsillar abscess (quinsy) is the most common deep-space infection of the head and neck tissues in children. It results from direct contiguous spread of infection from the tonsils. Older children and adolescents are most commonly affected without seasonal preference. Children with peritonsillar abscess have sore throat, neck pain, pain with swallowing or dysphagia, and fever. Physical exam reveals enlargement of the cervical lymph nodes, uvular deviation, and muffled ("hot potato") voice. Treatment options include broad-spectrum antibiotics, needle aspiration of the abscess, incision and drainage, and tonsillectomy. Possible complications are extension of the infection, acute upper airway obstruction, and rupture of the abscess with aspiration of purulent material.

6. Infectious Mononucleosis

Infectious mononucleosis is a triad of fever, lymphadenopathy, and tonsillar pharyngitis. It may lead to acute upper airway obstruction due to the enlargement of tonsils and adenoids. Fortunately, this complication, which tends to occur in younger children, is rare. Oral or parenteral corticosteroids may be helpful. Tracheal intubation or NIV may be required.

7. Miscellaneous Disorders

a. Airway Trauma

Traumatic injury to the upper airway may be caused by foreign bodies, thermal or chemical injury, or direct blunt or penetrating trauma. Foreign body aspiration is a leading cause of accidental death and severe neurologic impairment in young children. Infants are at significant risk of foreign body aspiration due to their tendency to put any and all objects into their mouths. Many aspirated foreign bodies pass through the vocal cords and become lodged in the distal airways. Any foreign body that becomes lodged in the larynx is life-threatening. A high index of suspicion is always necessary in that an actual aspiration event is frequently not identified. Children with a history of choking and respiratory distress should undergo immediate rigid bronchoscopy for diagnosis and treatment.

Life-threatening airway obstruction may develop as a result of inhalation injury, laryngeal burns, or caustic ingestions. Any child with a scald injury to the face or neck should be evaluated by bronchoscopy for potential inhalational injury. Inhalation injury should be suspected in children with evidence of soot in sputum or vomitus, singed nasal hairs, facial burns, lip burns, wheezing, stridor, or extensive surface burns. Early endoscopic evaluation is helpful for determining the presence and extent of airway injury. Management includes early tracheal intubation because airway burns tend to worsen with edema for several days before they begin to resolve (**Chapter 10**).

Blunt injury to the neck can cause edema and hematoma formation, resulting in acute upper airway obstruction. Penetrating injury is less common in children. Imaging and surgical consultation are necessary.

b. Post-extubation Airway Obstruction

Patients who are intubated sometimes develop signs and symptoms of airway obstruction upon extubation, referred to as *post-extubation stridor*. This condition is related to several factors, including preexisting tracheal irritation from gastroesophageal reflux disease, an inappropriately sized tracheal tube, traumatic intubation, the duration of intubation, global body edema, and a loosely secured endotracheal tube. Post-extubation stridor is often self-limited, resolving over 12 to 24 hours, but can also require intervention. Reintubation is necessary in some cases. Assessment and treatment is similar to that of viral croup. Dexamethasone may be administered both as treatment and as pre-extubation prophylaxis if post-extubation airway obstruction is anticipated. Repeated episodes of airway obstruction requiring reintubation and prolonged stridor after extubation are indications for bronchoscopy.

c. Angioedema

Angioedema is an immunologically mediated acute edema that may result in acute upper airway obstruction. It may affect the larynx; tissues of the head, neck, face, and lips; tongue; and pharynx. Angioedema most often occurs as an allergic reaction to food or medication, upper respiratory infection, or insect sting. IV corticosteroids, antihistamines, and intramuscular (IM) epinephrine (adrenaline) usually will bring about rapid improvement in symptoms. If medical interventions do not result in improvement or are not working rapidly enough, the airway should be secured in the presence of progressive signs and symptoms of obstruction.

d. Spasmodic Croup

Spasmodic croup is probably an allergic disease, not caused by infection. The infant typically wakes with sudden onset of respiratory distress, inspiratory stridor, and a loud, croupy cough. There may be a mild intercurrent upper respiratory illness and often a family history of asthma or allergies. The episodes may recur on successive nights. By the time the patient reaches the hospital, symptoms have usually resolved. The family often reports that symptoms abated in the cool night air. Dysphagia, drooling, high fever, and a toxic appearance are all absent in spasmodic croup.

IV. PHYSICAL FINDINGS AND ASSESSMENT

A. History

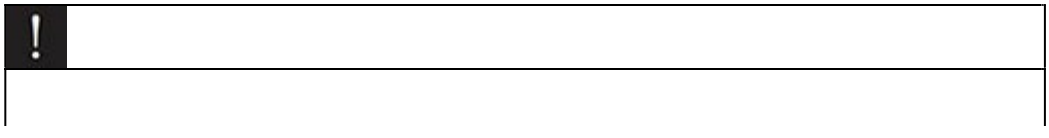
A brief history and careful physical exam will provide much of the information necessary to reach a diagnosis. Emergent intervention must take precedence when a child appears in severe distress from airway obstruction.

The duration of symptoms and their rapidity of onset are key areas on which to focus. Children with viral croup often have symptoms of upper respiratory infection for ≥1 days prior to the onset of stridor. Airway obstruction due to foreign body aspiration has an almost instantaneous onset. Epiglottitis has an acute onset, with fever and distress appearing without a prodrome. Retropharyngeal and parapharyngeal abscesses typically cause upper airway obstruction after several days of sore throat and related symptoms. A history of intubation in the neonatal ICU may precede a presentation of subglottic stenosis.

Additional points to cover in a history include: whether distress is stable or worsening; whether symptoms are intermittent or persistent; whether positional changes affect symptoms; presence or absence of fever; history of trauma or exposure to allergens, smoke, chemical vapors, or steam; changes in voice quality; report of choking episode; and possible foreign body aspiration.

B. Observation and Appearance

The degree of distress and severity of airway obstruction can often be ascertained by observation. Audible stridor indicates turbulent airflow through narrowed passages, but airflow is nonetheless present. Infants and young children with partial upper airway problems may appear calm, whereas older children and adults with partial obstruction will be agitated and extremely fearful. Some children, especially those with chronic airway obstruction, appear surprisingly at ease considering the difficulty they have breathing. Observe for accessory muscle use. Children who have been breathing against partially obstructed airways for several years often develop visible hypertrophy of accessory muscles of inspiration.



The degree of distress and severity of airway obstruction can often be ascertained by observation.

Children with acute airway-related processes often adopt a position of comfort to maximize airway patency and airflow. It is important to appreciate this positioning and attempt to maintain it. For example, a child who seems most comfortable in a caregiver's arms should not be removed. The most comfortable position is usually upright, and a patient may vigorously resist being moved out of this position. Moving children out of the position of comfort may induce unnecessary fear and worsen the degree of airway obstruction. As noted previously, increasing respiratory effort against an upper airway obstruction will result in tracheal collapse and worsening of obstruction. Thus, a patient with signs and symptoms of upper airway obstruction should be kept as calm as possible. Sedating a child before intubation or positioning a patient to facilitate laryngoscopy may result in complete airway obstruction.

Children with acute airway-related processes often adopt a position of comfort to maximize airway patency and airflow.

A tripod position, in which a child sits upright and leans forward on outstretched arms, is a sign of upper airway obstruction. By leaning forward, the patient tries to relieve the obstruction by using gravity to move enlarged tonsils, redundant pharyngeal tissue, or an edematous epiglottis forward and away from the airway, thus opening an air passage. A component of the tripod posture is the forward jutting of the jaw, which is the patient's attempt to move the pharyngeal contents anteriorly.

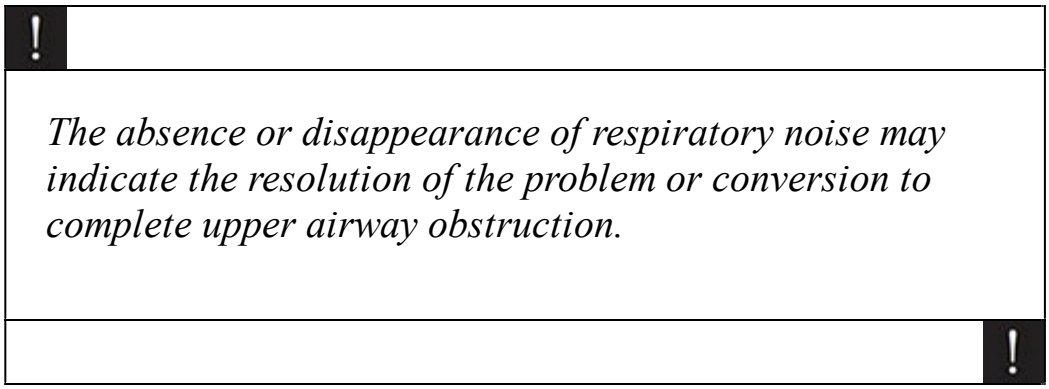
Sedating a child with partial upper airway obstruction before intubation or positioning a patient to facilitate laryngoscopy may result in complete airway obstruction.

In laryngomalacia, the epiglottis is long and soft and closes in over the laryngeal inlet with inspiration. Given that the epiglottis is an anterior component of the larynx, placing an infant in a prone position permits gravity to move the epiglottis out of the airway. An infant with laryngomalacia may breathe more easily when moved from a supine position to a prone position.

A listless appearance may indicate fatigue and signify impending respiratory or cardiac arrest. Observe for cyanosis. Listen for the quality of cough or voice, as it can guide the diagnosis.

C. Physical Examination

The physical exam should focus on the quantity and quality of airflow. Inspiration and expiration are normally quiet. Noise, accessory muscle use, and an appearance of unease indicate the presence of a pathologic situation. Inspiratory noise that emanates from the nose or pharynx is called stertor. Partial nasal obstruction, tonsillar-adenoidal hypertrophy (infectious and noninfectious), and peritonsillar and parapharyngeal abscesses may all cause this sound. Noise cannot occur without air movement. The absence or disappearance of respiratory noise may indicate resolution of the abnormality or conversion to complete upper airway obstruction.


!
The absence or disappearance of respiratory noise may indicate the resolution of the problem or conversion to complete upper airway obstruction.
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1. Inspiratory and Expiratory Noises

Inspiratory noise is characteristic of airway disorders from the nose to the subglottic, but extrathoracic, space. The presence of an expiratory component indicates that the process extends to the airways of the chest as well. For example, viral laryngotracheobronchitis may affect the airway from the level of the subglottic space (extrathoracic) to the bronchi (intrathoracic). The edema and mucous accumulation associated with severe croup may thus cause both inspiratory stridor from involvement of the extrathoracic airway and an expiratory component from involvement of the intrathoracic airways. Laryngomalacia involving the epiglottis, arytenoid cartilages, and laryngeal structures is extrathoracic and causes inspiratory noise. Tracheomalacia, in which the tracheal tissue is prone to collapse, may involve both intrathoracic and extrathoracic parts of the trachea. The extrathoracic trachea is prone to collapse with inspiration, whereas involved intrathoracic areas are prone to collapse with expiration, thus accounting for a characteristic biphasic stridor seen in severe tracheomalacia.

Stridor is a raspy, high-pitched noise that is caused by air moving through narrowed passages of the supraglottic, glottic, and subglottic spaces. It occurs most commonly with inspiration but may occur during expiration. It is most often associated with viral croup, subglottic stenosis, epiglottitis, and post-extubation airway edema. Stridor may be present at rest or during agitation.

When airway edema is treated with nebulized racemic epinephrine (adrenaline), stridor may disappear and then return when the medication wears off.

Children and infants who are large enough to move sufficient amounts of air often manifest a characteristic barking or seal-like cough with viral croup. Sufficient airflow is required for a cough to be present, and thus absence of cough is not necessarily reassuring. Small infants with viral croup may not manifest a classical croupy cough.

Peritonsillar abscess is associated with a "hot potato voice," which occurs due to pain and edema in the peritonsillar tissues that make it difficult to enunciate clearly.

Noisy stertorous sounds are usually associated with obstruction in the nose or retropharynx. In patients who can open their mouths, it may be possible to see obstructing tonsillar tissue or an exudative pharyngitis associated with infectious mononucleosis. It is unwise to persist at trying to open the mouth of a child who resists doing so. A tongue blade should not be used to examine the pharynx of a child with suspected epiglottitis because of the risk of converting a partial upper airway obstruction into a complete obstruction.

Auscultation over the nares can determine the presence or absence of airflow. High-pitched sounds over the throat indicate narrowing of the air passage. During auscultation of the chest, careful attention should be paid to whether adventitious sounds are inspiratory, expiratory, or both, and to which component is louder. Sounds related to the trachea and bronchi will be louder centrally, whereas small, distal airway-related sounds will be more prominent in the peripheral chest.

2. Fever

The presence of fever depends on the etiology of the airway disease. Congenital and acquired disorders, such as laryngomalacia and subglottic stenosis, are not associated with fever. Viral laryngotracheobronchitis is infectious in etiology, and a low-grade fever may be present. High fevers are usually associated with bacterial infections of the airway, such as parapharyngeal abscess, epiglottitis, and bacterial tracheitis.

D. Measurements and Imaging

Pulse oximetry is neither threatening nor painful and is useful in assessing a patient with acute upper airway disease. Acceptable oxygen saturation indicates that even in the presence of clinically visible distress, gas exchange is sufficient to provide an adequate amount of oxygen to the bloodstream. A drop in oxygen saturation may suggest a worsening of obstruction or the development of muscle fatigue and impending respiratory arrest.

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Although an arterial blood gas analysis can yield important and valuable information about a patient with upper airway disease, this is offset by the discomfort and difficulty of obtaining an arterial blood sample, compounded by the agitation that drawing it induces

Upper airway diseases do not usually affect the regions of the lungs responsible for gas exchange, but if airway obstruction limits gas flow to the extent that inadequate oxygen reaches the alveoli, the patient will manifest desaturation. Another mechanism of desaturation is ventilation-perfusion (V/Q) mismatch from atelectasis that develops when unrelieved inspiratory effort against an obstruction leads to respiratory muscle fatigue. The work of breathing and the oxygen consumption necessary to support the diaphragm and accessory muscles of inspiration can be tremendous when a patient breathes against a partially obstructed airway. The inability to meet the demand for oxygen in the face of inadequate gas flow ultimately results in anaerobic metabolism and lactic acidosis.

Although an arterial blood gas analysis can yield important and valuable information about a patient with upper airway disease, this is offset by the discomfort and difficulty of obtaining an arterial blood sample, compounded by the agitation that drawing it induces. One of the prime directives of managing a patient with upper airway obstruction is to limit noxious stimuli, and blood gas measurement in the absence of an indwelling arterial line is noxious.

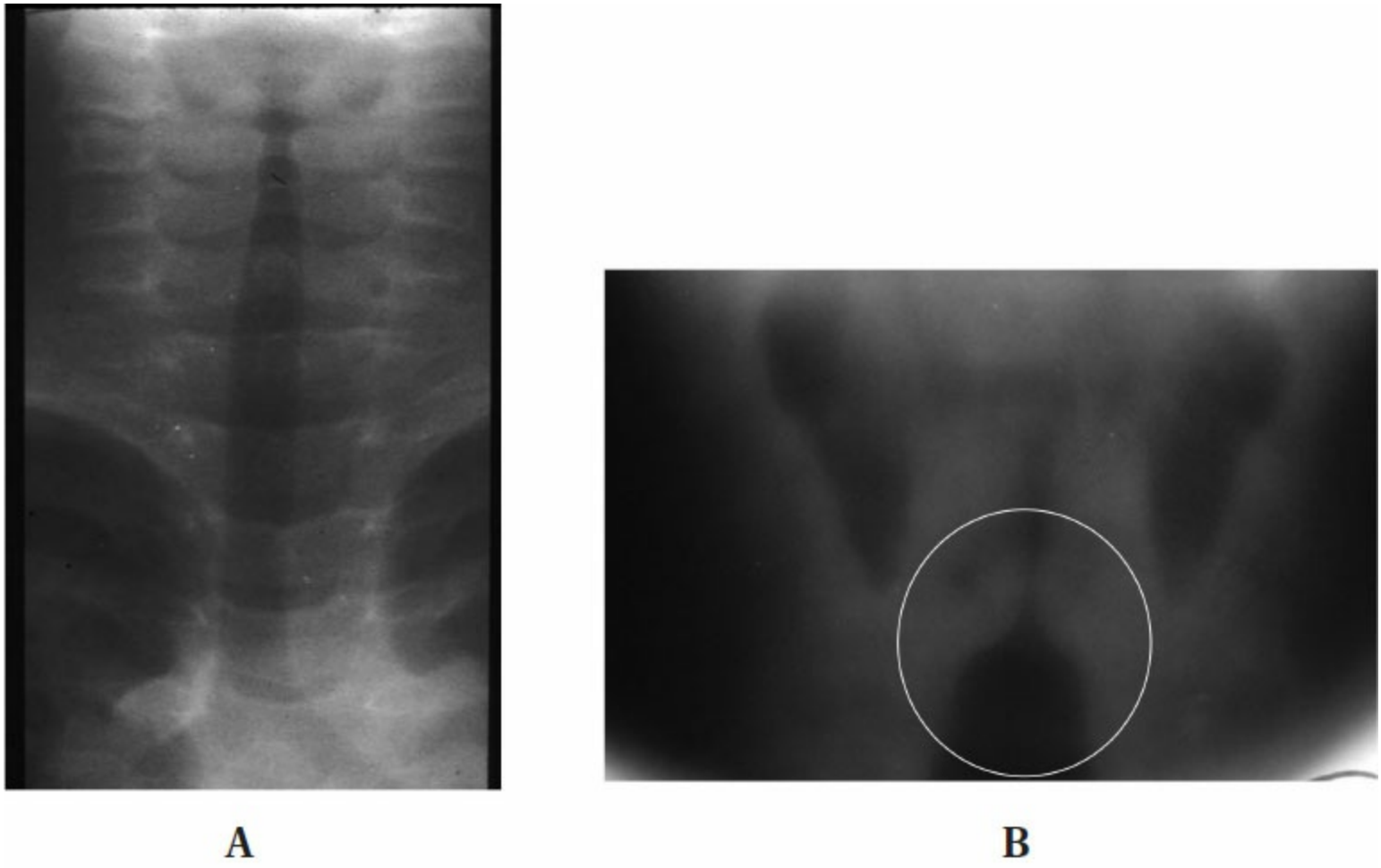
Sidestream end-tidal CO_2 sampled via nasal cannula can be helpful in assessing ventilation adequacy in a patient with upper airway obstruction. However, the necessary devices are not widely available.

Radiographic imaging, when safe for a patient with a compromised airway, can yield helpful information. Anteroposterior and lateral neck views with filtering to gain increased soft tissue resolution may show:

- Narrowing in the subglottic space (steeple sign) consistent with viral croup (**Figure 4-3**)
- Radio-opaque foreign bodies
- Enlarged, edematous epiglottis (thumb-print sign) consistent with epiglottitis (**Figure 4-4**)
- Hypertrophied adenoidal and tonsillar tissue
- Abnormally wide retropharyngeal or prevertebral tissues consistent with mass or with retropharyngeal or parapharyngeal abscess (**Figure 4-5**)

For comparison, films of a patient with normal airway anatomy are shown in **Figure 4-6**. Plain view radiographs of the chest can reveal a deviated or narrowed tracheal air column, compressed or narrowed bronchi, or the presence of atelectasis or pneumonia. More advanced imaging of the airways using fluoroscopy and computerized tomographic scanning with 3D reconstruction can also yield important information. Patient stability and the maintenance of the airway must be given top priority in planning all radiographic imaging.

Figure 4-6. Normal Airway



A, An anteroposterior radiograph of a normal tracheal airway shows a widely patent air column with some narrowing at the level of the vocal cords. **B**, An airway tomogram demonstrates a widely patent tracheal air column to the level of the vocal cords (circled) and the typical "shoulders" seen in a normal airway.
Courtesy of Terry L. Levin, MD, Children's Hospital at Montefiore Medical Center, New York, New York, USA

For a distressed patient with an upper airway obstruction, the safest and most effective approach to diagnosis begins with monitored transfer to surgery or the ICU. Skilled personnel in these locations can perform a careful examination of the airway using direct laryngoscopy or bronchoscopy. A multidisciplinary approach to the management of upper airway obstruction should be the standard operating procedure in any facility providing care for children.

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For distressed patients of all ages with upper airway problems, the safest and most effective approach to diagnosis begins with monitored transfer to surgery or the ICU.

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Several clinical scoring tools are available for assessing the severity of croup. The Westley croup scoring system has been extensively studied and validated. The scoring rubric (**Table 4-6**) is based on the presence and extent of inspiratory stridor, retractions, air entry, and cyanosis, and on the

patient's mental status. A Westley croup score lower than 3 suggests mild disease, a score of 3 to 6 indicates moderate disease, and a score higher than 6 represents severe disease.

Table 4-6	WESTLEY CROUP SCORE					
			(Points)			
Finding	**0**	**1**	**2**	**3**	**4**	**5**
Stridor	None	With agitation	At rest			
Retractions	None	Mild	Moderate	Severe		
Air Entry	Normal	Mild decrease	Marked decrease			
Cyanosis	None				With agitation	At rest
Level of Consciousness	Normal					Depressed
3 points: Mild disease						
4-6 points: Moderate disease						
>6 points: Severe disease						

V. MANAGEMENT OF ACUTE UPPER AIRWAY DISEASE

Patients with upper airway obstruction will appear frightened, anxious, and uncomfortable. The degree of interventions should be proportional to the level of distress and severity of the patient's clinical condition. For example, an infant with mild to moderate viral croup is comfortable in the caregiver's arms with blow-by humidified oxygen and can tolerate oral intake; arterial blood gas analysis, extensive blood testing, or an IV or fluid is not needed. In upper airway disorders, anxiety and agitation may worsen a patient's clinical condition. Interventions should be carried out in progressive steps from least to most invasive. In general, it is best to do the least, keeping in mind that definitive action may be required in some circumstances and cannot be delayed.

Children should be allowed to find a position of comfort that maximizes their ability to maintain a patent airway. Attempt to accomplish any interventions while patients are in their preferred position. Children with epiglottitis are often in their caregivers' arms, positioned with their chins up and jutted forward in a spontaneous jaw-thrust position. This will maintain a patent airway in the presence of a swollen epiglottis. One general approach to such patients is to keep them in their caregivers' arms until they are brought to the operating room for mask induction and intubation.

Attempt to accomplish any interventions while patients are in their preferred position.

Find a position that maximizes airflow and minimizes obstruction in a patient with a decreased level of consciousness. An infant with disorders of the chin and tongue, such as Pierre Robin sequence, may have a completely obstructed airway when lying supine, but is able to breathe easily in a prone position. Align the oral, pharyngeal, and tracheal axes in a spontaneously breathing patient to maintain airway patency. The airway axes can be lined up by placing a small roll under the shoulders of an infant or under the occiput of an older child, followed by careful neck flexion to place the patient in the sniffing position (**Chapter 2**). Monitor the changes in airflow through the nose and mouth or watch the oxygen saturation to evaluate the efficiency of these maneuvers. Patients with severe tonsillar-adenoidal hypertrophy are usually able to move air more easily in an upright position.

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In sedation-induced obstruction, opening the airway by means of chin lift, jaw thrust, or a combination of both will be more effective than supplemental oxygen in reversing oxygen desaturation.

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Partial upper airway obstruction due to backward movement of the tongue and reduced hypopharyngeal tone occurs frequently in children with a decreased level of consciousness. It may be seen in a child who has sustained a closed head injury, is recovering from surgical anesthesia, or is undergoing procedural sedation. In the latter situation, administration of supplemental oxygen will delay desaturation by replacing the patient's functional residual capacity with oxygen, but oxygen alone will not be effective in the presence of airway obstruction. In sedation-induced obstruction, the patient's airway can be opened using a chin-lift or jaw-thrust maneuver, which is more effective in improving oxygen saturation than supplemental oxygen. After the airway is opened, oxygen saturation will usually rise quickly provided the patient is not apneic. Maintaining airway patency by means of chin-lift and jaw-thrust maneuvers is a skill that all healthcare providers caring for seriously ill children should possess. Additionally, oropharyngeal and nasopharyngeal tubes can also be employed to maintain the airway.

A. Medical Interventions

1. Heliox

Helium-oxygen mixtures, known as heliox, are extremely useful when a mechanical or anatomic obstruction limits airflow. Heliox comes in 60:40, 70:30, and 80:20 helium-oxygen percentage mixtures. It can be administered by nonrebreather face mask or nasal cannula. A significant improvement in comfort is often seen following heliox therapy. The oxygen concentration is not high; in an 80:20 mixture, it is actually below the oxygen concentration of room air. Thus, heliox alone may not be adequate if the oxygen requirement is significant due to parenchymal lung disease. Oxygen can

be added, but fractional oxygen percentages above 40% will rapidly undo the benefits of heliox.

Heliox is effective because it is less dense than either oxygen or air and supports a more laminar airflow. When airways are narrowed by edema, compression, or anatomic abnormality, the gas flow becomes turbulent due to abrupt changes in velocity. In croup, turbulence is caused by constriction in the subglottic space. Heliox has its greatest affect in conditions affecting the larger airways.

Heliox is a very light, low density gas owing to the same properties that make it work for airway problems. The use of a tight-fitting, nonrebreather face mask and secure connections will ensure adequate delivery.

2. Racemic Epinephrine (Adrenaline)

Racemic epinephrine (2.25% by nebulizer), frequently used as a topical vasoconstrictor in the management of stridor, is usually effective in postextubation stridor and croup. The effects are transient and only meant to temporize until steroids or other measures provide longer lasting reduction in edema. For children younger than 4 years, the dose is 0.05 mL/kg mixed with 3 mL normal saline and administered by nebulizer every 2 to 4 hours. For children 4 years and older, the dose is 0.5 mL mixed with 3 mL normal saline administered every 2 to 3 hours (maximum dose, 0.5 mL every 1 to 2 hours). Onset of action can be expected in 1 to 5 minutes, and the effect may last 1 to 3 hours. Tachycardia can be pronounced, and continuous cardiorespiratory and pulse oximetry monitoring should be used. Although racemic epinephrine is usually prescribed for croup, it can be used for any upper airway disorder in which tissue edema may be a contributing factor. Recurrent stridor may ensue in 2 to 3 hours and has been reported as a reason for hospital admission in children who receive racemic epinephrine in the emergency department. The half-life of racemic epinephrine may be too short to clear the subglottic narrowing of viral.

There is little information about the use of continuous nebulized racemic epinephrine, but if a patient with a complicated airway is showing a clinical response, continuous nebulized treatments may be helpful.

3. Dexamethasone

Dexamethasone administered orally or parenterally has been shown to be effective in viral laryngotracheobronchitis. The literature has suggested a dose of 0.6 mg/kg given once. For postextubation airway edema, the dosing is 0.5 to 2 mg/kg/day IM or IV divided every 6 hours. Dexamethasone given 24 hours before extubation and continuing for 4 to 6 doses afterwards can be used to prevent postextubation stridor.

4. Noninvasive Ventilation (NIV)

NIV may be useful in cases of upper airway obstruction. The addition of positive pressure to the airways can decrease the amount of extrathoracic tracheal collapse that occurs when a patient attempts to inhale against a partial obstruction. Those with obstruction due to tonsillar-adenoidal hypertrophy can experience significant relief through the use of NIV. Although this pressure may be beneficial, it may also force secretions down into the trachea and inhibit their clearance.

In cases of severe inspiratory difficulty, complete airway obstruction, or respiratory arrest, the patient can usually be supported by means of bag-mask ventilation. Effective ventilation may be difficult to achieve with obstruction. Healthcare providers must maintain an open airway. The 2-person bag-mask ventilation technique may be employed (**Chapter 2**); one person can ensure the mask is tightly sealed on the patient's face while the other can compress the bag to ensure adequate chest rise. The addition of a positive end-expiratory pressure valve to the bag-mask ventilator prevents end-exhalation airway collapse and may facilitate ventilation.

Direct laryngoscopy should be considered in a patient suspected of foreign body aspiration who has not responded to basic life support.

Racemic Epinephrine
2.25% administered by nebulizer
Age <4 years: 0.05 mL/kg mixed with 3 mL normal saline every 2-4 hours
Age ≥4 years: 0.5 mL mixed with 3 mL normal saline every 2-3 hours
Maximum dose: 0.5 mL every 1-2 hours
Onset of action: 1-5 minutes
Duration of action: 1-3 hours
Possible rebound: 2-3 hours

Dexamethasone
Viral croup: 0.6 mg/kg (PO, IM, IV) once (maximum dose 8-20 mg)
Postextubation stridor: 0.5-2 mg/kg/day IM or IV divided every 6 hours; start 24 hours before extubation and continue for 4-6 doses after extubation

VI. ACUTE LOWER AIRWAY DISEASE

Case Study

A 5-year-old girl with a history of asthma since she was 2 years old is brought to the emergency department with worsening tachypnea and wheezing despite frequent albuterol inhalation treatments at home. Her vital signs are as follows: heart rate, 135 beats/min; respiratory rate, 40 breaths/min; and pulse oximetry, 89% on room air. She appears anxious. On auscultation she has bilateral inspiratory

and expiratory wheezes with intercostal retractions. She is given an albuterol nebulizer treatment followed by oxygen via face mask. You are asked to assist in her management.

Detection

– What is the diagnosis?

– What is her physiologic state?

– Is she in respiratory distress or respiratory failure?

Intervention

– What are the next interventions?

Reassessment

– How do you assess her response to therapy?

– How should the patient be monitored?

Effective Communication

– What instructions will be given to the primary care team with respect to signs of worsening clinical condition?

– For what should they call you back to reassess the patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Respiratory failure is the inability of the pulmonary system to meet the metabolic needs of the body. The pulmonary system is involved in two crucial metabolic roles: elimination of CO_2 and oxygenation of the blood. There are three forms of respiratory failure: hypoxemic, hypercapnic, and mixed. Hypoxemic respiratory failure is defined by a room air Pao_2 ≤60 mm Hg (≤8.0 kPa). It may be caused by:

- V/Q mismatch
- Decreased diffusion of oxygen across the alveolocapillary membrane
- Alveolar hypoventilation
- High altitude or other causes of low inspired oxygen tension

Hypercapnic respiratory failure is defined by $Paco_2$ ≥50 mm Hg (>6.7kPa) with an accompanying acidosis (pH <7.35). It may be caused by:

- Decreased tidal volume
- Decreased respiratory rate
- Increased physiologic dead space
- Increased CO_2 production, which rarely produces significant hypercapnia

In both of these types of respiratory failure, hypoxemia is present to some degree. The diagnosis of respiratory failure by arterial blood gas analysis may not be readily available at the first encounter with the ill child, so the clinician may need to rely on the clinical indicators (**Table 4-7**).

Table 4-7	CLINICAL INDICATORS OF RESPIRATORY FAILURE

- Marked tachypnea (respiratory rate >60 breaths/min abnormal at any age)
- Bradypnea and/or apnea
- Cyanosis
- Worsening oxygen saturations despite intervention
- Tachycardia or bradycardia
- Poor or absent distal air movement
- Lethargy, stupor, and coma

VII. MANAGEMENT OF RESPIRATORY FAILURE

The initial goal of the management of respiratory failure is the restoration and stabilization of ventilation and oxygenation. Interventions should focus on a rapid assessment of the type and severity of respiratory compromise rather than on the precise etiology. An action plan with direct management should follow, emphasizing the need for frequent reassessment of the response to treatment and the planning of further interventions as needed.

The most vital aspect of management is securing the patient's airway. While attempting to establish an airway, it may be necessary to assist ventilation with a bag-mask device (**Chapter 2**). Oxygen saturation, end-tidal CO_2, and cardiovascular monitoring should be implemented.

VIII. CAUSES OF ACUTE RESPIRATORY FAILURE

Acute respiratory failure develops in a variety of clinical settings. In children, it typically results from diseases affecting the upper airway, lower airway, and lung parenchyma. All etiologies of upper and lower respiratory disease discussed in this chapter can cause respiratory failure. Diseases affecting other organ systems, including the cardiovascular and central nervous systems, may result in acute respiratory failure without respiratory distress.

1. Pathophysiology of Asthma

Asthma is an inflammatory disease that is manifested by air flow obstruction. This obstruction results from narrowing of the small and intermediate airways as a result of both bronchospasm (due to airway hyperresponsiveness) and mucosal edema with mucous plugging. Asthma is the most common chronic disease of childhood, is a leading cause of school absence, and accounts for a growing number of hospitalizations each year. Asthma exacerbations can be triggered by inhaled irritants (cigarette smoke), exercise, gastroesophageal reflux, allergen exposure, respiratory viral infections, and psychological stress. Airway obstruction results from the triad of smooth muscle spasm, mucosal inflammation, and mucous plugging. The obstruction and increased airway resistance caused by bronchoconstriction and airway edema impede airflow (expiratory greater than inspiratory), resulting in air trapping, hyperinflation, and V/Q mismatch. Pulmonary mechanics are further hampered by hyperinflation and flattening of the diaphragms. Expiration becomes an active process, requiring the use of accessory muscles. The increased work of breathing increases tissue oxygen demand which cannot be satisfied in the presence of hypoxemia from V/Q mismatch. These factors combine to result in tissue hypoxia, respiratory muscle fatigue, and respiratory failure.

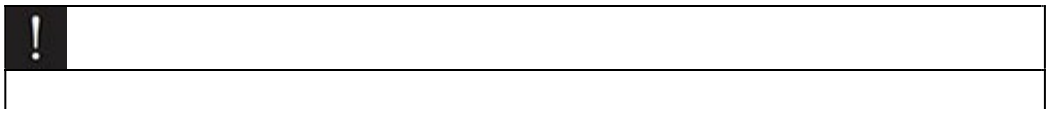
2. Clinical Findings and Diagnosis of Asthma

Cough, dyspnea, and wheezing are the major clinical features in asthma, but the presentation varies with age. In some children, the presentation is persistent cough at night and during exercise. In others, shortness of breath may be the predominant symptom. In infants, the first asthmatic episode is frequently associated with viral infection. In older children, episodes are usually preceded by symptoms of upper respiratory tract infection (ie, rhinorrhea and coughing followed by wheezing). During acute exacerbation, the cough usually sounds tight and is generally not productive. The degree of wheezing does not correlate well with the severity. In fact, the relative absence of wheezing in the presence of respiratory distress and poor air entry on lung auscultation is indicative of severe airflow obstruction. The use of accessory muscles of respiration, an active expiratory phase, and the presence of pulsus paradoxus signify severe compromise of respiratory function. As the condition worsens, patients begin trapping air and take on a barrel-chested appearance.

Children with severe acute asthma usually present with tachypnea, hyperpnea, retractions, and nasal flaring. These patients appear anxious; they may prefer a tripod sitting position, cannot be distracted from their breathing, and are typically unable to speak in full sentences.

a. Laboratory and Radiographic Tests

The routine complete blood count is usually normal and rarely useful in the assessment of acute asthma. Elevated blood counts usually reveal eosinophilia and leukocytosis; the latter may be induced by stress or corticosteroid administration. Therefore, an elevated white blood cell count does not always signify the presence of infection.



A silent chest with very limited airflow is a worrisome finding in children with asthma.

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A chest radiograph is indicated when the patient responds poorly to bronchodilators. The radiograph can identify any associated parenchymal disease, complications (eg, pneumothorax), and foreign bodies. It frequently shows lung hyperinflation and peribronchial thickening. Small segmental areas of atelectasis are frequently observed during the acute exacerbation and may be misinterpreted as pneumonia.

Typical blood gas findings in an acute uncomplicated asthma attack reveal low Pao_2, low $Paco_2$, and respiratory alkalosis. Hypoxemia is caused by V/Q mismatch, and low $Paco_2$ is due to hyperventilation. Normalization of the $Paco_2$ in a patient with worsening airway obstruction should be interpreted as a sign of respiratory muscle fatigue and impending respiratory failure. Hypoxemia is a more ominous finding in acute asthma and requires rapid intervention. Patients with respiratory acidosis with or without metabolic acidosis should be monitored closely in an ICU. Hypercapnia alone is not an indication for intubation if a patient is responding to therapy and remains conscious and hemodynamically stable. Conversely, normocapnic patients who exhibit hypoxemia, unconsciousness, or hemodynamic instability require immediate intubation and ventilator support.

b. Differential Diagnosis of Asthma

Congenital Malformations. Anomalies of respiratory, cardiovascular, and gastrointestinal systems can cause varying degrees of airway obstruction that may mimic asthma. Among the most common are laryngotracheobronchomalacia, vocal cord paralysis, tracheal or bronchial stenosis, lobar emphysema, lung cysts, vascular rings, and gastroesophageal reflux.

Foreign Bodies. Sudden onset of dyspnea, cough, and respiratory distress in previously healthy children with no history of recurrent episodes is suggestive of a foreign body in either the trachea or bronchial tree. Aids to diagnosis include a high degree of suspicion, differential sounds on chest auscultation, and a chest radiographs in inspiratory, expiratory, and lateral decubitus positions.

Croup. The prevalence of recurrent croup is increased in children with asthma, as is the incidence of persistent airway hyperreactivity of both the upper and the lower respiratory tract in children with recurrent croup.

Acute Bronchiolitis. Syndromes caused by RSV and other respiratory viruses occur in the first 2 years of life. Clinical signs include cough, coryza, wheezing, and progressive dyspnea. The condition may progress to respiratory failure. These syndromes are difficult to differentiate from asthma. Chest radiographs often show evidence of hyperinflation resulting from generalized bronchiolar obstruction, patchy pulmonary infiltrates, and atelectasis.

3. Management of Asthma

a. Corticosteroids

Corticosteroids are a mainstay in the management of acute and chronic asthma. Glucocorticoids suppress cytokine production, granulocyte-macrophage colony-stimulating factor, and inducible nitric oxide synthase activation, all of which are components of the inflammatory processes of asthma. Methylprednisolone is the most common corticosteroid used in critically ill asthma patients. An initial dose of 2 mg/kg is followed by 0.5 to 1 mg/kg/dose IV every 6 hours. Corticosteroids begin to exert their effect in 1 to 3 hours and reach a maximal effect in 4 to 8 hours. The duration of therapy is dictated by the clinical response. Consider gradually tapering the agents in patients requiring more than 5 to 7 days of steroid therapy.

b. Inhaled β-Agonists

β-Agonists cause direct bronchial smooth muscle relaxation and are key components of acute and chronic asthma therapy. Albuterol (salbutamol) is recommended at a dose of 0.05 to 0.15 mg/kg every 20 minutes for 3 doses. Continuous albuterol nebulization is effective and should be considered in patients failing to respond to intermittent treatments. The recommended dose for continuous albuterol is 0.15 to 0.45 mg/kg/h, with a maximum dose of 20 mg/h. The recommended levalbuterol dose is 0.075 mg/kg (minimum dose of 1.25 mg) every 20 minutes for 3 doses, followed by 0.075 to 0.15 mg/kg (not to exceed 5 mg) every 1 to 4 hours as needed. It is important to monitor serum potassium levels in patients undergoing prolonged continuous inhalational therapy due to intracellular potassium shifts that can result in hypokalemia. Frequent administration of high-dose inhaled β-agonists in patients who are dehydrated often results in severe tachycardia that can be ameliorated by judicious fluid boluses.

c. Fluids

Critically ill children with status asthmaticus are often dehydrated as a result of decreased oral intake prior to admission and a rise in insensible fluid losses from increased minute ventilation (tachypnea). Providing appropriate fluid resuscitation and ongoing maintenance fluid is essential. The increased intrathoracic pressure generating by air trapping will decrease venous return to the right side of the heart. This underfilling is further worsened by the effects of β-agonists via tachycardia and relative diastolic hypotension. This issue can become critical if the patient is started on NIV or mechanical ventilation.

d. Anticholinergic

Ipratropium bromide is the most frequently used anticholinergic agent in the treatment of asthma. It promotes bronchodilation without inhibiting mucociliary clearance. It can be delivered either by aerosol or metered-dose inhaler. Dose range is 125 to 500 μg (nebulized) or 4 to 8 puffs administered every 20 minutes for up to 3 doses. The subsequent recommended dosing interval is every 4 to 6 hours.

e. Magnesium Sulfate

Magnesium acts as a bronchodilator primarily through its activity as a calcium channel blocker and its role in activation of adenylate cyclase in smooth muscle cells. As a result, magnesium inhibits

calcium-mediated smooth muscle contraction and facilitates bronchodilation. The usual dosing of magnesium is 25 to 50 mg/kg/dose IV over 30 minutes, administered every 4 hours. It can also be given by continuous infusion at a rate of 10 to 20 mg/kg/h. Hypermagnesemia and related muscle weakness are rare within the prescribed dosing range.

f. Intravenous and Subcutaneous β-Agonists

IV and subcutaneous administration of β-agonists is beneficial in children with severe status asthmaticus in whom reduced respiratory air flow may limit distribution of inhaled medications. Terbutaline is a relatively selective β_2-agonist. Subcutaneous administration is primarily used for patients without IV access and as a rapidly available adjunct to an inhaled β-agonist. The subcutaneous dosing for terbutaline is 0.01 mg/kg/dose with a maximum dose of 0.3 mg. The dose may be repeated every 15 or 20 minutes for up to 3 doses. IV terbutaline is also useful in severe status asthmaticus and is started with a loading dose of 10 μg/kg over 10 minutes, followed by a continuous infusion of 0.1 to 1 μg/kg/min. It is rare that a dose higher than 4 μg/kg/min is needed. As with inhaled β-agonists, monitoring of potassium levels in important.

g. Methylxanthines

Methylxanthines promote relaxation of bronchial smooth muscle. The exact mechanism remains controversial, although suggested mechanisms include increasing intracellular cyclic adenosine monophosphate levels by blocking phosphodiesterase 4, control of calcium influx, inhibition of endogenous calcium release, and prostaglandin antagonism. Though not recommended in the National Heart, Lung, and Blood Institute acute asthma management guidelines, theophylline therapy can be helpful in critically ill children who have an inadequate response to steroids and β-agonist therapy. Aminophylline and theophylline, though often effective in very sick patients, have complicated pharmacokinetics and serious side-effect profiles. They should be used with careful attention to the complexities of their pharmacology.

Aminophylline is administered by continuous IV infusion following a loading dose of 5 to 7 mg/kg infused over 20 minutes. Continuous infusion should begin immediately after the loading dose at a rate of 0.5 to 0.9 mg/kg/h. Serum theophylline levels should be measured 2 to 4 hours after the loading dose and then 6 to 8 hours after initiation of continuous infusion. The pharmacokinetics of aminophylline is usually consistent, and an average volume of distribution of 0.5 L/kg can often be reliably used to predict serum levels from dosing. However, at serum levels greater than 20 to 25 μg/mL (20 to 25 mg/L), pharmacokinetics can become zero order and thus levels are not in a fixed relationship to dose. For maximal therapeutic benefit, the goal is a theophylline serum level of 10 to 20 μg/mL (10 to 20 mg/L). All patients on continuous IV infusion of β-agonists or aminophylline should be monitored by continuous cardiac monitoring.

h. Heliox

Heliox in helium-oxygen concentrations of 80:20 and 70:30 may be useful as adjuvant therapy in severe asthma. Due to its low density, heliox can potentially improve the work of breathing by facilitating laminar gas flow in areas of high airway resistance and help in the delivery of albuterol. The use of heliox is recommended in patients who are not hypoxemic and who fail to respond to

conventional treatments. The low fraction of inspired oxygen in heliox mixtures may limit its use in hypoxemic patients. Heliox is not considered a standard therapy in the management of asthma.

i. Noninvasive Ventilation

NIV has been shown to decrease the work of breathing and improve air movement in adults and children with asthma. By opening edematous narrowed airways and limiting expiratory airway collapse, NIV can reduce the work of breathing, reduce distress, and improve hypoxemia. It may reduce the need for intubation and mechanical ventilation in some patients. It is a reasonable intervention for patients who have dyspnea and signs of increased work of breathing (intercostal and subcostal retractions). NIV provides almost instant reduction in the work of breathing, which results in it being generally well tolerated by children. Both intermittent and continuous nebulized medications can be administered while the patient is receiving NIV.

A nasal or full face mask that fits well is absolutely necessary. Patients who are not severely distressed may feel better even with a nasal mask. Initial settings with a peak pressure of 10 to 12 cm H_2O and positive end-expiratory pressure (PEEP) of 5 to 6 cm H_2O are reasonable for initiation of therapy. Settings may be titrated by bedside examination. A blood gas should be obtained 30 to 60 minutes after initiation to assess the efficacy.

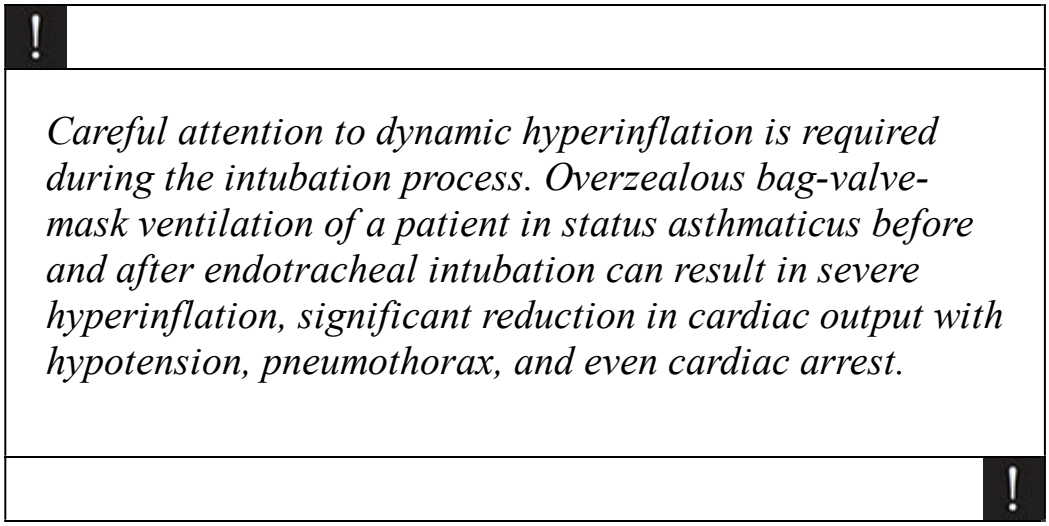
j. Invasive Mechanical Ventilation

Though efforts are always made to minimize tracheal intubation in pediatric asthma patients, intubation is absolutely indicated at times and should not be delayed. Tracheal intubation is indicated for children following cardiorespiratory arrest, as well as those with refractory hypoxemia and hypercarbia (not responsive NIV), severe respiratory and metabolic acidosis, and depressed mental status. Mechanical ventilation is intended to provide a stable airway, oxygen delivery, and ventilation adequate to reduce severe acidosis while the underlying pathology resolves. Most importantly, mechanical ventilation helps alleviate the extreme work of breathing that occurs in status asthmaticus.

The goals of ventilatory support in status asthmaticus should be to maintain adequate oxygenation and allow for permissive hypercarbia (moderate respiratory acidosis) by adjusting minute ventilation (peak pressure, tidal volume, and rate) to maintain pH >7.2.

Ventilator management strategies should minimize dynamic hyperinflation from air trapping. Utilizing a low breath rate with a long expiratory phase and short inspiratory time (low inspiratory-expiratory ratio) will ensure that expiratory airflow returns to zero before initiation of the next inspiration, thus minimizing air trapping. Using ventilator graphics in addition to physical exam to insure termination of exhalation before initiation of the next breath can help minimize dynamic hyperinflation. Judicious use of PEEP (5 to 10 cm H_2O) can facilitate expiratory airflow by limiting expiratory airway collapse. Efforts to normalize Pco_2 by increasing ventilator rates usually result in even higher Pco_2 values and intrathoracic pressures as this results in worsened air trapping

Once the patient is intubated and placed on a ventilator, efforts should be made to minimize the use of non-depolarizing neuromuscular blockade agents. These agents greatly increase the risk of severe and prolonged myopathy which can occur when the muscle relaxants are used simultaneously with high-dose corticosteroids.


!
Careful attention to dynamic hyperinflation is required during the intubation process. Overzealous bag-valve-mask ventilation of a patient in status asthmaticus before and after endotracheal intubation can result in severe hyperinflation, significant reduction in cardiac output with hypotension, pneumothorax, and even cardiac arrest.
!

k. Inhaled Anesthetics

Inhaled anesthetics, such as isoflurane and sevoflurane, act as direct bronchodilators. They can be considered in situations in which patients fail to respond to conventional therapy. Coordination with the anesthesiology department regarding equipment and management is essential.

l. Extracorporeal Membrane Oxygenation

In severely refractory cases of asthma, extracorporeal membrane oxygenation (ECMO) can be applied. Overall survival to discharge for patients with asthma requiring ECMO support is 84% to 92%. Venovenous ECMO may be performed if the patient has adequate cardiac output. Hypercarbia can be rapidly resolved, and care must be taken to moderate the rate of CO_2 reduction to avoid cellular shifts and cerebral edema. Rapid improvement is the rule, and most patients require 2 to 5 days of ECMO support.

4. Intubation in Severe Status Asthmaticus

Patients with asthma not only have severe respiratory compromise, but are also at risk for emergent cardiovascular alterations. Intubation is often judiciously avoided given potential complications. Most patients undergoing intubation are unstable and have little cardiopulmonary reserve. The cosegregation of increased intrathoracic pressure from air trapping, decreased diastolic blood pressure from β-agonist exposure, hypovolemia from dehydration, and increased insensible losses can lead to sudden cardiovascular collapse. All necessary resuscitation medications should be drawn and ready, and fluid boluses should be given before intubation. This situation is further compromised by use of sedation and neuromuscular blocking agents, as this results in reduction of the patient's endogenous adrenergic tone and vascular competency. The use of cardiovascularly neutral medications, such ketamine or etomidate, may also help in support of these difficult patients. Ketamine is preferred for its bronchodilatory effects; succinylcholine or rocuronium may be used for paralysis. True rapid sequence induction should be attempted, but if a bag-valve-mask device is required, ventilation rates should be kept at 8 to10 breaths per minute to avoid further air trapping. These patients are also at risk of an air leak syndrome (pneumothorax) during intubation.

B. Bronchiolitis

Case Study

A previously healthy 1-month-old boy, born at 34 weeks' gestation, is brought to the emergency department by his mother. She reports that the infant developed a runny nose and some cough about 1 week ago. His symptoms have not improved despite nebulized albuterol treatments every 6 hours. During the last 12 hours, he has begun grunting, flaring, and retracting. He appears less active and has been feeding poorly. The mother is concerned because the infant stops breathing intermittently.

Detection

– What is in the differential diagnosis?

– What is the likely diagnosis?

– What tests would be useful to evaluate the severity of the patient's condition?

Intervention

– What pharmacologic treatment should be initiated?

Reassessment

– How should the patient be monitored and in what setting?

– Intermittent apneas continue; are there other interventions to try?

Effective Communication

– For what do you want to be called back?

– What do you want to tell the nursing team about the patient?

Teamwork

– Who is going to do what and when?

– How will the treatment plan be implemented?

1. Pathophysiology of Bronchiolitis

Bronchiolitis is an acute inflammatory disease of the lower respiratory tract that results in obstruction of small airways. It is the most frequent cause of apnea after hospital discharge in patients younger than 2 months. RSV, human metapneumovirus, rhinovirus, and coronavirus are the most common causes and tend to occur in epidemics during winter and spring. Bronchiolitis may also occur with other viruses, including adenovirus, influenza, and parainfluenza.

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The diagnosis of bronchiolitis must be considered in neonates presenting with apnea after hospital discharge.

Preterm infants, those with cyanotic and noncyanotic congenital heart disease, infants with bronchopulmonary dysplasia, and children who are immunodeficient or immunosuppressed are susceptible to bronchiolitis and tend to have severe manifestations of the illness.

2. Clinical Manifestations of Bronchiolitis

The infant is typically exposed to an adult or child with an upper respiratory tract infection. Initial symptoms include cough, sneezing, rhinorrhea, and often low-grade fever. Subsequently, patients develop tachypnea, nasal flaring, retractions, wheezing, and irritability. Cyanosis may be reported. Thoracoabdominal asynchrony correlates with the degree of airflow obstruction. Lung examination reveals diffuse wheezes, prolonged expiration, and rales. Apnea occurs both as a presenting symptom and later in the illness.

3. Diagnosis of Bronchiolitis

Appropriate tests for patients with severe bronchiolitis include a chest radiograph, complete blood count, rapid fluorescent antibody test for RSV and influenza, and polymerase chain reaction for common viruses, if available. In most infants with bronchiolitis, the chest radiograph demonstrates hyperinflation due to air trapping, and patchy infiltrates are common. Often, the radiograph reveals new atelectasis in an infant with bronchiolitis who has a sudden decompensation. The right upper lobe is commonly affected in this fashion and may be mistaken for a lobar infiltrate by practitioners not familiar with this finding. The complete blood count is usually normal.

4. Management of Bronchiolitis

a. Hydration and Oxygen

Hydration and oxygen comprise the backbone of care for an infant with bronchiolitis. Oral hydration should be attempted, but if the patient is unable to tolerate this because of respiratory distress, nasogastric feeding or IV fluids can be considered. Supplemental oxygen (nasal cannula or face mask) should be used to maintain oxygen saturations above 92%. Humidification of supplemental oxygen is mandatory to prevent desiccation of airway secretions.

b. Naso-oropharyngeal Suctioning

As most infants are obligate nose breathers, clearance of the nares and pharynx can do much to alleviate the respiratory distress of bronchiolitis. Simple airway suctioning reduces airways resistance, increases oxygenation and ventilation, and avoids compromise of the patient's functional residual capacity.

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Secretions can be copious in bronchiolitis, and careful attention to suctioning of the nose and airways can be beneficial.

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c. β_2-Agonists

Inhaled β-agonists have no proven benefit in bronchiolitis; however, in a clinical setting, it remains common practice to administer them by nebulizer at least on a trial basis. If clinical improvement is not noted or if worsening is observed, these agents should be discontinued.

d. Racemic Epinephrine (Adrenaline)

Racemic epinephrine has been studied, and a trial (2.25% solution: 0.05 mL/kg/dose diluted in 3 mL of saline via nebulizer) may be warranted. Discontinue if no improvement is observed.

e. Inhaled and Systemic Steroids

The use of inhaled and systemic steroids is controversial, as these agents have not been proven to have any effect in acute bronchiolitis. When the distinction between viral bronchiolitis and recurrent wheezing due to asthma is difficult to discern, steroids may be tried as a therapeutic intervention.

f. Intubation and Mechanical Ventilation

A small number of affected patients will have respiratory failure severe enough to prompt intubation. Mechanical ventilation management can be challenging in infants with bronchiolitis as the disease can manifest as poor compliance parenchymal disease and as obstructive airway disease. Good pulmonary toilet is essential in the care of the intubated patient. Sudden clinical decline should raise the suspicion of bacterial superinfection.

C. Pneumonia

1. Pathophysiology and Classification of Pneumonia

Pneumonia is an inflammation of the lung parenchyma caused by bacterial or viral infection. It is characterized by a reduction in total lung capacity that is associated with a decrease in compliance. Expiratory flow rates are usually preserved, and major obstructive defects are rare. Progressive alveolar involvement and deterioration of gas exchange lead to respiratory distress and failure.

Pneumonia can be classified by the anatomic location (lobar, lobular, alveolar, or interstitial), environment where it was acquired (community or nosocomial), or causative organism. Although a universal definition is lacking, community-acquired pneumonia is usually described as a lung

infection with acute symptoms (fever, cough, dyspnea), associated with altered pulmonary auscultation (crackles) or the presence of an acute infiltrate on the chest radiograph of a previously healthy child. Pneumonias acquired after 48 to 72 hours of hospitalization are considered nosocomial, which should inform empiric antibiotic selection.

When a child presents with recurrent pneumonia, the possibility of an underlying disease or anatomic anomaly (eg, acquired or congenital lung anatomic abnormalities, immunodeficiency, tracheoesophageal fistula, foreign body, cystic fibrosis, heart failure, untreated asthma, bronchiectasis, ciliary dyskinesia, neutropenia, and increased pulmonary blood flow) should be considered.

2. Diagnosis of Pneumonia

The diagnosis of parenchymal lung disease is challenging. Auscultative findings may be completely normal unless significant consolidation or small airways disease (fine crackles late in inspiration) exist. A chest radiograph is critical for both the diagnosis and the management of pneumonia. Radiographs may help identify lobar versus diffuse infiltrates and can aid in the identification of complications, such as pleural effusion or pneumothorax.

Fever and cough are frequently present, and clinical signs of tachypnea, accessory muscle retractions, and altered pulmonary auscultation tend to be the most specific indicators of lower airway compromise. Usual signs and symptoms of pneumonia are fever, lethargy, poor appetite, pallor or cyanosis, toxemia or toxic appearance, and abdominal distension. Right lower lobe pneumonia has been mistaken for appendicitis. Signs of lung compromise include intercostal and subcostal retractions, nasal flaring, and thoracic pain. Findings on auscultation vary and may include alterations in breath sounds, such as bronchophony and crackles.

3. Management of Pneumonia

a. Supportive Therapy

Supportive therapy includes: maintenance of the nutritional state; fluid, electrolyte, and acid base balance; humidified oxygen therapy; thinning of secretions; and physiotherapy. Tracheal and mechanical ventilation is necessary in severe cases with respiratory failure.

b. Antibiotic Therapy

Group B streptococci and Gram-negative bacteria prevail among newborns until the age of 3 weeks; in most of the cases, IV ampicillin and gentamicin should be used. In severe disease, a third-generation cephalosporin (cefotaxime) should be used, along with ampicillin to provide coverage for potential *Listeria monocytogenes*. In children between 4 months and 4 years of age, ampicillin at 200 mg/kg/d divided and given every 6 hours should be administered. Ceftriaxone should be used in severe cases. In selecting antibiotics, careful attention must be given to local bacterial resistance patterns.

In children older than 5 years, a 5-day course of azithromycin can be used for routine treatment of atypical bacteria, particularly *M pneumoniae*. Recommended dosing is 10 mg/kg on day 1, followed

by 5 mg/kg/d for 4 subsequent days. Ceftriaxone, with or without macrolides, may be used in acutely ill children. At all ages, if the clinical manifestations suggest the presence of *S aureus*, oxacillin or vancomycin should be used, depending on the prevalence of methicillin-resistant staphylococcus in the community. Staphylococcal superinfection is frequently associated with influenza pneumonia.

c. Noninvasive Ventilation

NIV should be utilized for patients who remain hypoxemic despite high oxygen concentrations. Indications for intubation in pneumonia are respiratory failure with severe hypoxemia, worsening muscle fatigue, and altered mental status. If endotracheal intubation is necessary, consider adding a PEEP valve to the bag-valve-mask device before intubating, as PEEP will improve oxygenation and recruit collapsed alveoli.

4. Complications

Complications of pneumonia include pleural effusions, empyema, extrapulmonary infections and sepsis, acute respiratory distress syndrome, shock, lung abscess, pneumothorax, atelectasis, and multiple organ system dysfunctions. Pleural effusion and empyema are the most frequent complications.

Pleural effusions often accompany bacterial pneumonia in pediatric patients. *Streptococcus pneumoniae* is the most commonly associated organism, accounting for 22% of cases in the United States. *Staphylococcus aureus* and *Streptococcus pyogenes* are also associated with a high incidence of pleural effusion and empyema. Parenchymal necrosis appears to be an increasingly common complication of pediatric bacterial pneumonia.

Chest radiographs, sometimes with right and left lateral decubitus views, are used to diagnose pleural effusions, assess their size, and determine if they are free flowing. Increasingly, ultrasound of the chest is used to assess the specific locations of effusions and whether or not they contain fibrinous strands, which contribute to loculations that make thoracentesis drainage difficult. Empyema is distinguished from a parapneumonic effusion in that it is purulent with a high white blood cell count and a low pH. Empyemas also tend to be less free flowing with a greater incidence of loculation.

Treatment options for effusions and empyema include provision of supplemental oxygen and/or antibiotics, supportive therapy plus pleural drainage via thoracostomy tube with or without intrapleural thrombolytic infusion, and supportive therapy plus aggressive pleural drainage using techniques such as video-assisted thoracoscopic surgery. The decision to perform thoracostomy drainage in pediatric patients with a parapneumonic effusion may depend on the clinical context in which it occurs. Pronounced compromise of lung volume or delayed clinical improvement may inform this decision. In cases of significant pleural fluid organization, some favor the administration of intrapleural thrombolytics to facilitate evacuation of fluid through the chest drain; studies assessing the efficacy of this practice have produced conflicting results. Video-assisted thoracoscopic surgery may facilitate chest drainage through direct visual inspection of the pleural space, mechanical disruption of adhesions, and placement of chest drains in strategic locations.

D. Cystic Fibrosis

Case Study

A 17-year-old girl with cystic fibrosis comes to the emergency department with a 1-week history of increased cough, wheezing, and changes in the quantity and quality of her sputum. She has had chest pain and increased work of breathing since this morning. Her current oxygen saturations of 89% are considerably lower than her baseline value.

Detection

– What are possible explanations for her clinical condition?

– What diagnostic studies should be obtained?

– What tests would be useful to evaluate the severity of the patient's condition?

Intervention

– What treatment interventions should be instituted?

Reassessment

– With what frequency should this patient's condition be reassessed?

– What clinical indicators should be followed?

Effective Communication

– Who should be informed of this patient's condition?

– What should the inpatient staff be told about this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is doing what and when?

1. Pathophysiology of Cystic Fibrosis

Cystic fibrosis (CF) is an autosomal recessive disorder involving a mutation on the long arm of chromosome 7 that causes abnormal chloride and sodium ion transport across epithelial surfaces, resulting in viscid secretions that lead to the blockage of airways. The lower airway is normal at birth but will develop obstructive pulmonary disease over time as a result of recurrent airway inflammation, chronic mucus production, and recurrent infections. Children usually present with recurrent coughing and wheezing, bronchiolitis, asthma, and pneumonia. They eventually develop hyperinflation with chronic diffuse bronchiectasis. Pneumothorax occurs in about 10% of patients and can be a common cause of chest pain and respiratory failure.

2. Diagnosis of Cystic Fibrosis

Patients with CF tend to have upper airway involvement with pansinusitis and nasal polyps. The bacteria responsible for exacerbations early in the disease process may include *S aureus*, *H influenzae*, and *Klebsiella* species. *Pseudomonas aeruginosa* (mucoid strain), *Aspergillus fumigatus*, *Burkholderia cepacia*, and *Stenotrophomonas maltophilia* become the predominant organisms later in the process.

Sweat testing should be considered in patients with:

- History of meconium ileus with prolonged jaundice in the neonatal period
- Rectal prolapse, chronic diarrhea with steatorrhea
- Nasal polyps and pansinusitis
- Chronic coughing and wheezing
- Documented *S aureus* pneumonia or documented *Pseudomonas* in sputum culture
- Failure to thrive
- Digital clubbing
- Family history of CF

3. Management of Cystic Fibrosis

Respiratory failure is relatively uncommon in children with CF. However, it may occur in undiagnosed children presenting with viral or bacterial pneumonia, in patients with advanced CF presenting with pneumonia or an acute pneumothorax, or in terminally ill CF patients presenting with right heart failure.

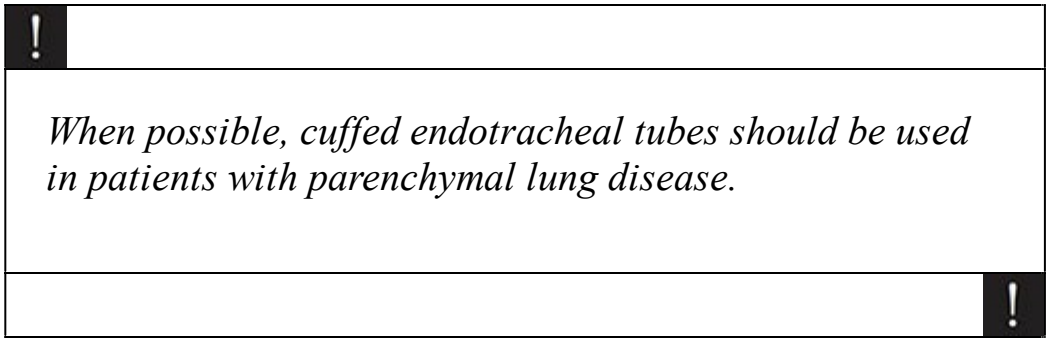
A patient with CF may benefit from:

- Chest physical therapy and postural drainage
- Inhalation therapy with bronchodilators
- Mucolytic agents (*N*-acetylcysteine, 3-5 mL of 20% solution via nebulizer 3 times daily)
- Recombinant human DNase
- Vasoconstrictors

Antibiotic treatment may vary from a short course of one antibiotic for uncomplicated cases to a longer term course of multiple antibiotics in more complex cases. Treatment initially should be directed against *Pseudomonas* species. Recommended IV therapy includes cefepime (150 mg/kg/d) or ceftazidime (150 mg/kg/d) in combination with tobramycin (7.5 mg/kg/d). Vancomycin, 45 mg/kg/d IV, should be considered in patients with suspected staphylococcal infections. Early consultation with a pediatric pulmonologist should be sought when available.

1. Definition and Pathophysiology of Acute Respiratory Distress Syndrome

The acute respiratory distress syndrome (ARDS) is manifested as acute noncardiogenic pulmonary edema with bilateral pulmonary infiltrates on a chest radiograph. The syndrome is associated with significant hypoxemia. The diagnosis and severity rating used for children originally were based on adult criteria. In 2015, specific definitions for pediatric acute respiratory syndrome (PARDS) were developed and published by the Pediatric Acute Lung Injury Consensus Conference Group (**Table 4-8**). These criteria define pediatric ARDS as a specific disease process and set standard descriptions for use in research in pathophysiology, therapeutics, and prognostics.



ARDS can develop following either direct or indirect lung injury. Pneumonia and pulmonary aspiration commonly cause direct lung injury and can result in ARDS, but traumatic pulmonary contusion, fat embolism, submersion injury, and inhalation injury are relatively common causes as well. The most common forms of indirect lung injury include systemic conditions such as sepsis, shock, cardiopulmonary bypass, and transfusion-related lung injury. Direct injury is suspected of causing regional consolidation from destruction of the alveolar architecture, while indirect injury is believed to be associated with pulmonary vascular congestion, interstitial edema, and less severe alveolar involvement.

Table 4-8 PEDIATRIC ACUTE RESPIRATORY DISTRESS SYNDROME, 2015 PEDIATRIC ACUTE LUNG INJURY CONSENSUS CONFERENCE GROUP

Age	Exclude patients with perinatal lung disease
Timing	Within 7 days of known clinical insult
Origin of edema	Not explained by left cardiac failure or fluid overload
Chest imaging	Findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease
Oxygenation:	
Noninvasive ventilation	
	No risk stratification Full face mask bi-level ventilation or continuous positive airway pressure ≥ 5 cm H_20 PF ratio ≤ 300 (Pao_2/F_{IO_2}) SF ratio ≤ 264 (Sao_2/F_{IO_2})
Invasive mechanical ventilation (OI preferred to P/F or S/F, use OSI when OI not available)	
	Mild 4≤ OI >8, 5≤ OSI >7.5

	Moderate 8≤ OI >16, 7.5≤ OSI >12.3 Severe OI ≥16, OSI ≥12.3
Special Population	
Cyanotic heart disease	
	Standard criteria above for age, timing, origin of edema, with chest imaging with an acute deterioration in oxygenation not explained by underlying cardiac disease
Chronic lung disease	
	Standard criteria above for age, timing, and origin of edema with chest imaging consistent with new infiltrate and acute deterioration in oxygenation from baseline which meet oxygenation criteria above
Left ventricular dysfunction	
	Standard criteria above for age, timing, and origin of edema with chest imaging consistent with new infiltrate and acute deterioration in oxygenation which meet criteria above not explained by left ventricular dysfunction

Data from Pediatric Acute Lung Injury Consensus Conference Group. Pediatric acute respiratory distress syndrome: consensus recommendations from the Pediatric Acute Lung Injury Consensus Conference. *Pediatr Crit Care Med*. 2015 Jun;16(5):428-439.

OI = oxygenation index ($OI=FIO_2$ x mean airway pressure x $100/Pao_2$)

OSI = oxygen saturation index ($OSI= FIO_2$ x mean airway pressure x $100/Spo_2$

If using OSI, titrate oxygen to achieve $Spo_2<97\%$

ARDS progresses through states defined by associated clinical, radiographic, and histopathological features. The first, or exudative, phase is characterized by the acute development of decreased pulmonary compliance and hypoxemia. The alterations in pulmonary mechanics lead to tachypnea. Arterial blood gas analysis typically reveals hypercarbia, and the chest radiograph reveals diffuse alveolar infiltrates from pulmonary edema. The proinflammatory events that occur in the exudative phase create the setting for transition into the fibroproliferative stage, during which increased alveolar dead space and refractory pulmonary hypertension may develop as a result of chronic inflammation and scarring of the alveolar capillary unit. The fibroproliferative phase gives way to a recovery phase, with restoration of the alveolar epithelial barrier, gradual improvement in pulmonary compliance, resolution of arterial hypoxemia, and eventual return to premorbid pulmonary function in many patients.

2. Management of Acute Respiratory Distress Syndrome

The mainstay of therapy for ARDS is supportive care, of which a critically important component is positive pressure ventilation. The breakdown of the alveolar capillary barrier in ARDS, as well as surfactant dysfunction, lead to a severe decline in pulmonary compliance and lung volume, leading to ventilation and perfusion mismatch. This explains why hypoxemia in ARDS is usually refractory to supplemental oxygen alone and requires the application of positive pressure ventilation. A trial of NIV is warranted; however, in severe hypoxemia, invasive mechanical ventilation may be required as the initial mode of support. Strategies for mechanical ventilation in ARDS include aggressive efforts to reduce ventilator-associated lung injury by using low tidal volumes (as low as 4-6 mL/kg) to maintain ventilator plateau pressures $\leq$30 cm H_2O and reduction of administered oxygen by using PEEP aggressively to recruit and maintain patency of alveolar lung units. The ARDSNet study demonstrated improved outcomes in adult patients with ARDS who were treated with these

strategies; no similar studies have been done in pediatric patients.

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Mechanical ventilation is provided using a lung-protective strategy.
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Adjuvant therapies include prone positioning and corticosteroids. Alveolar consolidation occurs along the gravitational axis in acute lung injury and ARDS, and pulmonary blood flow is distributed preferentially to dorsal (dependent) lung regions. It is logical to speculate that the ventilation-perfusion ratio in the injured lung can be improved by changing body position. Studies have failed to show a decrease in mortality with prone positioning. Occasionally improvements in gas exchange may be substantial enough to allow the patient to wean from potentially injurious inflation pressure or high oxygen concentrations.

Corticosteroids limit transudation of plasma across the capillary endothelium and exert anti-inflammatory effects. Studies evaluating a short course of high-dose corticosteroids administered in early ARDS did not demonstrate any survival benefit.

F. Pulmonary Edema

1. Pathophysiology of Pulmonary Edema

There are 2 types of pulmonary edema: high-pressure pulmonary edema and permeability pulmonary edema. The former is frequently cardiogenic in origin, whereas the latter is typically caused by a disruption of the alveolocapillary membrane. The increase in capillary pressure associated with high-pressure pulmonary edema is caused by conditions that result in obstruction of blood flow into the left atrium (pulmonary venous obstruction) or through the left heart (impaired left ventricular function or mitral valve stenosis). Rising left atrial pressure due to impaired mitral valve function or poor left ventricular function is transmitted in a retrograde fashion through the pulmonary veins to the pulmonary capillary bed and increases the rate of fluid flux into the interstitial space surrounding the alveoli. This net flow overwhelms the ability of the lymphatic system to clear the fluid. Edema fluid can break through the tight walls of the alveolar epithelium and cause flooding in the alveolar air space, producing hypoxemia and a decrease in alveolar compliance. Peribronchial edema leads to closing of the small airways, decreasing pulmonary compliance further and increasing the work of breathing. Increased hydrostatic pressure is the primary mechanism by which interstitial and alveolar flooding occurs in cardiogenic pulmonary edema. Patients with low plasma protein are more likely to develop pulmonary edema at lower levels of left atrial pressure than those with normal oncotic pressure.

Permeability or noncardiogenic pulmonary edema occurs when the alveolar capillary membrane is damaged directly by an inhaled toxin or when pulmonary surfactant is inadequate in quantity or function. This is also seen with the capillary leak syndrome displayed in systemic inflammatory

response syndrome, anaphylaxis, and septic shock. There are no direct interventions for permeability pulmonary edema aside from judicious fluid management and positive pressure ventilatory support.

Postobstructive pulmonary edema sometimes occurs in patients whose severe upper airway obstruction has been relieved. It is seen in patients with severe obstructive sleep apnea who undergo tonsillectomy and adenoidectomy and who suddenly become hypoxemic postoperatively, as well as in patients with acute upper airway obstruction who are intubated and pink frothy fluid is subsequently suctioned from their tracheal tube.

2. Diagnosis of Pulmonary Edema

Children might present with only mild dyspnea and failure to thrive. With worsening symptoms, chest radiographs show cephalization of fluid, pulmonary congestion, and clearly evident Kerley B lines. The presence of frank pulmonary edema may be discovered only when postintubation suctioning produces pink frothy fluid.

3. Management of Pulmonary Edema

Patients with pulmonary edema may benefit from:

- A timely cardiology consultation if a cardiogenic, high-pressure etiology is suspected.
- Furosemide (1-2 mg/kg/d in divided doses every 6-12 hours) to reduce extracellular volume and decrease systemic venous return, by lowering atrial filling pressure and improving cardiac function. The acute relief in pulmonary congestion is probably related to a venodilatory effect. The drug also promotes transcapillary refill from the interstitial space. Diuresis should be approached cautiously in patients with shock.
- Bed rest with the head of the bed elevated at 30° to 45°.
- Reduction of pulmonary venous return (preload) and systemic vascular resistance (afterload).
- Morphine (0.1-0.2 mg/kg/dose) to reduce preload by increasing venous capacitance. Morphine decreases venous return and reduces total peripheral resistance.
- Reduction of myocardial metabolic demand via treatment of hyperthermia.
- Positive pressure ventilation, either NIV or invasive, with increased PEEP. This is more effective in easing the increased work of breathing and hypoxemia than supplemental oxygen.

Care should be used in administering large amounts of supplemental oxygen to patients with cardiogenic or high-pressure pulmonary edema because the reduction in pulmonary vascular resistance may worsen pulmonary edema by dilating the pulmonary venous system.

Endotracheal intubation should be considered in patients with persistent hypoxemia and respiratory failure despite maximum noninvasive supplementary oxygen support and in patients with hemodynamic compromise. The cardiovascular collapse noted in some patients after intubation and bag-valve ventilation may be the result of a decrease in venous return with positive pressure

ventilation. Additionally, measures designed to minimize the increase in intrathoracic pressure, such as decreasing inspiratory time, decreasing tidal volume, and optimizing the amount of PEEP, should be considered. Cardiopulmonary resuscitation may be necessary, and healthcare providers must always be prepared for this possibility.

G. Acute Chest Syndrome in Sickle Cell Disease

Acute chest syndrome (ACS) is an acute lung injury syndrome that occurs frequently in patients with sickle cell disease. This syndrome is defined as a new pulmonary infiltrate on a chest radiograph consistent with alveolar consolidation but not atelectasis, involving at least 1 complete lung segment. The radiographic findings are usually accompanied by chest pain, fever, tachypnea, wheezing, cough, or hypoxemia. Although often self-limited, some episodes progress rapidly to acute respiratory failure, causing high rates of morbidity and death.

The 3 mechanisms usually postulated as causing ACS are pneumonia or systemic infection, fat embolism, and direct pulmonary infarction from sickle hemoglobin-containing erythrocytes. Though patients with sickle cell disease are at increased risk of pulmonary thromboembolism, the role of pulmonary emboli in ACS has not been clarified. The systemic hypoxemia associated with a pulmonary process in sickle cell disease may serve to worsen sickling overall and especially in the affected lung areas. ACS may develop 1 to 3 days after hospitalization for sickle cell veno-occlusive disease, or it may be the cause for admission.

Useful therapeutic interventions include:

1. Adequate hydration while avoiding fluid overload
2. Oxygen for hypoxia
3. Antibiotics to treat the usual causes of bacterial pneumonia, as well as macrolide therapy
4. Analgesia sufficient to permit deep breathing and incentive spirometry
5. Simple red blood cell transfusion if hemoglobin is <10 g/dL
6. Partial or full volume exchange transfusion if severe symptoms persist when the hemoglobin is >10 g/dL
7. NIV if passive oxygen delivery is inadequate or if the work of breathing is significant

On most occasions, a substantial improvement in oxygenation and comfort is achieved with simple or exchange transfusion.

Key Points

Diagnosis and Management of the Child With Acute Upper and Lower Airway

Disease

- Children should be allowed to remain in their position of comfort as long as possible.
- Any intervention should focus on a rapid assessment of the type and severity of respiratory compromise rather than the precise etiology.
- Frequent reassessment of the response to treatment is vital for good outcome.
- Airway maneuvers, such as jaw thrust and chin lift, can reverse hypoxemia caused by upper airway obstruction resulting from decreased levels of consciousness.
- A nasopharyngeal tube can be lifesaving in patients with severe airway obstruction due to Pierre Robin sequence.
- A multidisciplinary team with expertise in medical and surgical airway management should be assembled for cases of suspected epiglottitis and other severe cases of upper airway obstruction.
- Direct examination of the airway in the operating room by skilled anesthesiologists and otolaryngologists is the correct way to diagnose severe upper airway obstruction. Radiographic examination should be reserved for children who are relatively stable and whose diagnosis is in question.
- Bag-mask ventilation can provide lifesaving oxygenation and ventilation even in cases of complete airway obstruction due to epiglottitis.
- Heliox is useful in all varieties of upper airway obstruction.
- Respiratory failure can exist with or without respiratory distress.
- In patients with asthma, the degree of wheezing is unrelated to the severity of the disease. A silent chest with very limited airflow is a worrisome finding.
- Cuffed endotracheal tubes should be used when a significant change in compliance is anticipated.

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MECHANICAL VENTILATION

Objectives

- Identify the indications, advantages, and disadvantages of noninvasive ventilation.
- Describe the distinguishing characteristics of the various mechanical ventilators and their modes.
- Select the appropriate mode of ventilation, ventilator settings, and monitoring parameters to initiate mechanical ventilation.
- Explain the relationship between parameters of mechanical ventilation and modifications needed to avoid its harmful effects.
- Briefly discuss approaches to weaning from mechanical ventilation.
- Outline the use of lung-protective strategies in patients with acute respiratory insufficiency and acute respiratory distress syndrome.

Case Study

A 6-year-old boy underwent thoracotomy and drainage of a pleural abscess 2 days ago. A left thoracostomy tube was left in place. The operative course was uneventful, and he was extubated on postoperative day 1 without difficulty. This morning, he is noted to have increased work of breathing and saturations of 92% on 6 L/min of oxygen via face mask. A chest radiograph shows complete atelectasis of the left lung. He is started on noninvasive ventilation (NIV) with the following settings: inspiratory positive airway pressure, 12 cm H_2O; expiratory positive airway pressure, 6 cm H_2O; and fraction of inspired oxygen (F_{IO_2}), 0.5 with a backup rate of 20. A repeat chest radiograph obtained 4 hours later shows near-complete re-expansion of the left lung.

Detection

– What was the most likely reason for the radiographic findings before the initiation of NIV?

– What are the advantages and disadvantages of NIV therapy in this setting?

Intervention

– How should the noninvasive ventilator settings be managed, and what tests or assessments are appropriate at this time?

Reassessment

– Is the current treatment strategy effective?

– Are there any other interventions that might be helpful to this patient?

– If the repeat chest radiograph did not show any improvement, what steps might have been taken to promote lung re-expansion?

Effective Communication

– What is the best clinical setting in which to manage the care of this patient?

– How will you communicate a change in status? Who needs to know the care plan? How will that information be disseminated?

Teamwork

– How will the treatment plan be implemented?

– Who is responsible for implementing ventilation changes?

I. INTRODUCTION

The recognition of and response to respiratory failure continues to be a top priority in the management of critically ill children. The anatomic considerations unique to the respiratory tracts of infants and children and the inherent lack of respiratory reserve during times of stress pose special challenges to the caretaker confronted with this urgent situation. Regardless, the goal is always the same: to restore and maintain adequate oxygenation and ventilation while optimizing patient comfort and avoiding further injury. Failure to accomplish this goal can lead to devastating consequences, including cardiopulmonary arrest and death.

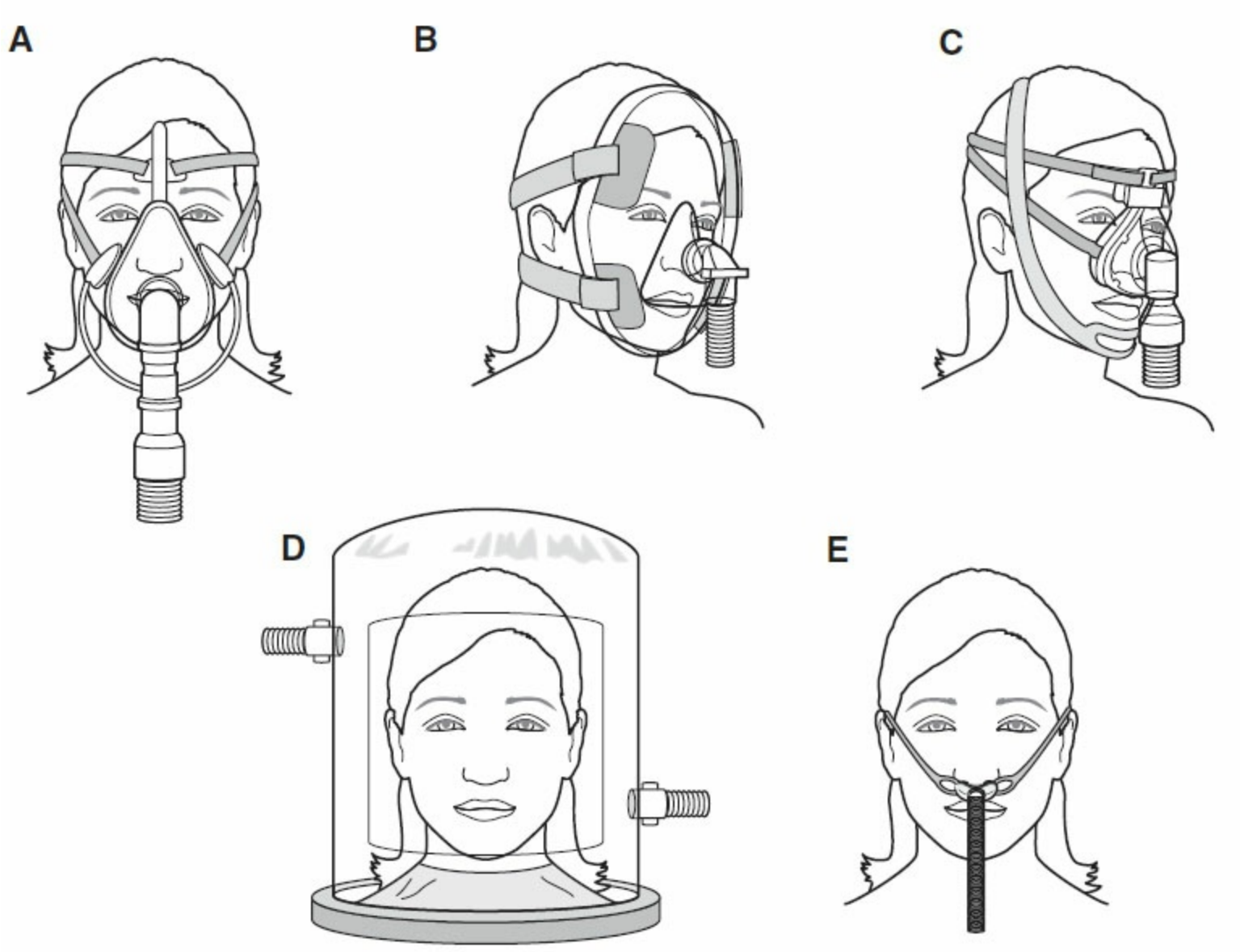
This chapter briefly outlines the general approach to the management of the child requiring mechanical ventilation, with a focus on the use of mechanical ventilation in specific clinical settings in the child with normal lungs or mild respiratory disease. Both conventional ventilation techniques and recent advances in respiratory support will be reviewed. Escalation to pediatric critical care is essential in children with more severe lung disease who require ongoing care.

II. NONINVASIVE VENTILATION

NIV can augment oxygenation and ventilation without insertion of an endotracheal airway. NIV is commonly described by the following terms: continuous positive airway pressure (CPAP),

noninvasive positive pressure ventilation, and bilevel positive airway pressure. These methods augment ventilation by delivering pressurized air through a nasal cannula, nasal or facial mask, or helmet. CPAP is not an actual mode of ventilation because no breaths are delivered if the patient is apneic. Younger patients may tolerate nasal devices best, but these can allow significant air leak via the patient's mouth. Mask devices must be fit well to avoid air leakage. Although a helmet-like apparatus may improve patient tolerance, such devices are not commonly used in pediatrics (**Figure 5-1**).

Figure 5-1. Devices for Delivery of Noninvasive Ventilation



A) face mask, **B**) total face mask, **C**) nasal mask with chin strap, **D**) helmet, **E**) nasal pillows

The delivery of positive inspiratory pressure is helpful in patients with chronic obstructive lung disease, neuromuscular disorders, obstructive sleep apnea, cystic fibrosis, upper airway obstruction, status asthmaticus, and acute chest syndrome. It has also been used as a means to avoid intubation in patients with mild to moderate acute respiratory insufficiency. NIV can significantly improve the respiratory rate, heart rate, work of breathing, and dyspnea score in the acute setting. It can be especially useful in previously healthy children with respiratory embarrassment due to muscle deconditioning following prolonged intubation.

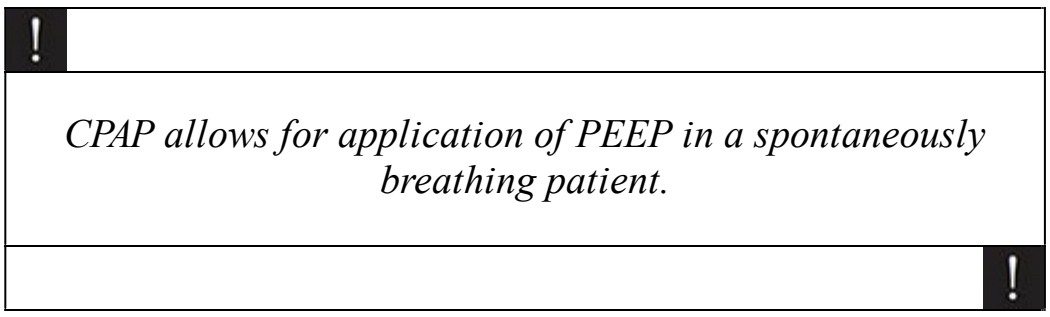
NIV is contraindicated in patients with:

- Rapidly progressive respiratory failure
- Hemodynamic instability, ie, cardiac arrest or shock
- Risk of aspiration
- Loss of protective airway reflexes or inability to clear copious oropharyngeal secretions
- Altered mental status
- Inability to properly fit the mask
- Periods of apnea
- After facial or upper airway surgery

NIV respiratory support requires the patient's cooperation. Sedation with a dexmedetomidine infusion or intermittent doses of midazolam may improve tolerance.

CPAP delivers a constant airway pressure throughout the respiratory cycle, similar to positive end-expiratory pressure (PEEP). The initial setting is usually 5 cm H_2O, titrated up as needed. This mode is dependent on the patient's own respiratory effort and rate. CPAP prevents the collapse of distal airways and alveoli, thus decreasing the work of breathing.

Bubble CPAP is useful in infants with respiratory insufficiency and preterm newborns with respiratory distress syndromes. Humidified oxygen is delivered via a nasal cannula or mask. Pressure in the circuit is maintained by immersing the distal end of the expiratory tubing in water. The depth of the tubing in water determines the amount of pressure delivered. This mode of NIV avoids the deleterious effects of an endotracheal tube in the developing trachea. This device is useful in resource-limited areas because full implementation does not employ extensive technology. Nevertheless, the bubble nasal CPAP requires minimal training to acquire the skills and experience necessary for beneficial clinical effects.



NIV adds an assisted ventilation pressure, which is either flow- or time-cycled. When the ventilator detects inspiratory flow, an inspiratory positive airway pressure is delivered, cycling to a lower expiratory positive airway pressure when inspiratory flow ceases or after a set time, to assist the patient's spontaneous effort. Initial settings are presented in **Table 5-1**. NIV can also be utilized to deliver other respiratory modes, including timed delivery of breaths, which can replace or supplement the patient's own rate; however, these modes are less frequently used in the pediatric

population.

Table 5-1	INITIAL SETTINGS FOR NONINVASIVE VENTILATION[a]
Age	**Initial Setting**
Infants <12 months	Nasal CPAP should be attempted first. If CPAP does not provide adequate support, endotracheal intubation is indicated.
Toddlers 1-2 years	IPAP, 8 cm H_2O; EPAP, 4 cm H_2O; F_{IO_2}, 1.0; ± backup rate appropriate for age and disease.
Children >2 years	IPAP, 10 cm H_2O; EPAP, 5 cm H_2O; F_{IO_2}, 1.0; ± backup rate appropriate for age and disease.

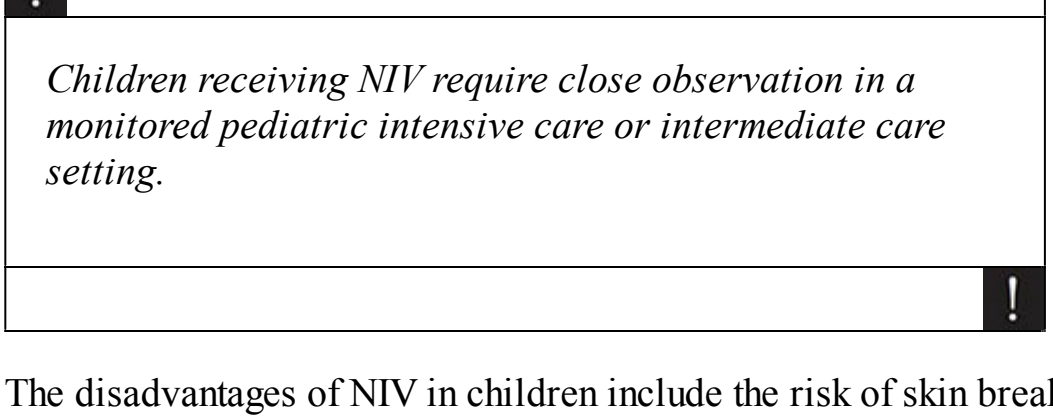
CPAP, continuous positive airway pressure; IPAP, inspiratory positive airway pressure; EPAP, expiratory positive airway pressure; F_{IO_2}, fraction of inspired oxygen.

[a] Maximum settings are dependent on patient and age, and effective delivery relies at least in part on the adequacy of the seal rather than the settings. If the patient can tolerate a peak inspiratory pressure >18-20 cm H_2O and positive end-expiratory pressure >12-15 cm H_2O, but the work of breathing is not decreased or oxygenation does not improve sufficiently, or if respiratory insufficiency is not improved, tracheal intubation is indicated.

Appropriate settings are based on the nature and severity of the patient's respiratory insufficiency. A word of caution: machines are often insufficiently sensitive to the respiratory efforts of small patients. Careful observation is required to prevent counterproductive or poorly timed mechanical breaths (ie, dyssynchrony).

Improvement of symptoms with initiation of NIV can be characterized by decreases in respiratory rate and/or heart rate, improvement in oxygenation, or resolution of hypercapnia. If these improvements are not observed in 1 to 2 hours, endotracheal intubation should be considered. A lack of improvement or continued decline may indicate a need for intubation earlier than this.

The advantages of NIV include decreases in the risks of laryngeal trauma, ventilator-associated pneumonia, tracheitis, and sedation and analgesia. In addition, if a nasal mask is utilized, the patient can talk, cough, and cooperate with pulmonary toilet.


!
Children receiving NIV require close observation in a monitored pediatric intensive care or intermediate care setting.
!

The disadvantages of NIV in children include the risk of skin breakdown over the nasal bridge and an increased risk of aspiration due to gastric distension. Masks may not fit tightly and may not provide effective ventilation, particularly in small patients. Restriction of activity is often needed to maintain optimal mask placement. Tracheal suctioning requires removal of the facial mask, which can lead to respiratory decompensation. Clinicians must closely monitor synchrony and triggering of the NIV,

especially in younger patients.

III. MECHANICAL VENTILATORY SUPPORT

A. Indications

Indications for endotracheal intubation are addressed in **Chapter 2**. When hypoxemic or hypercapnic respiratory failure cannot be treated by other means, the generally accepted indications for initiating mechanical ventilatory support may apply (**Table 5-2**).

Table 5-2	INDICATIONS FOR MECHANICAL VENTILATORY SUPPORT
Ventilation abnormalities	Respiratory muscle dysfunction • Respiratory muscle fatigue • Chest wall abnormalities • Neuromuscular disease Decreased ventilatory drive Increased airway resistance and/or obstruction
Oxygenation abnormalities	Refractory hypoxemia Need for positive end-expiratory pressure, as in pulmonary edema or pulmonary hemorrhage Excessive work of breathing

Mechanical ventilation is also indicated for the following purposes:

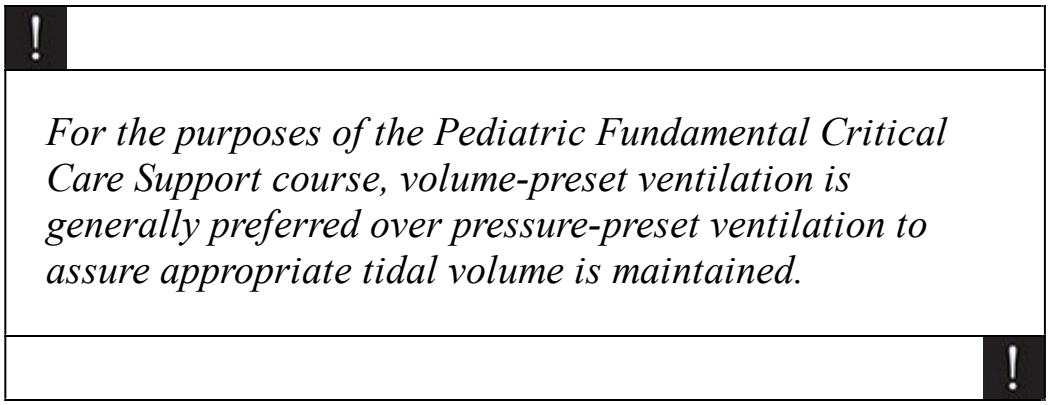
- To permit moderate to deep sedation and/or neuromuscular blockade
- To decrease systemic and myocardial oxygen consumption
- To allow transient hyperventilation for critically elevated intracranial pressure
- To facilitate alveolar recruitment and prevent atelectasis
- To improve oxygen delivery in conditions associated with inadequate oxygen supply/demand states (shock, sepsis, cardiopulmonary arrest)
- To prevent aspiration in patients with abnormal airway protective reflexes

B. Ventilator Variables

1. Phase Variables

Mechanical ventilators and their modes can be distinguished by the phase variables—trigger, limit,

and cycle—that govern the characteristics of the inspiratory phase of each breath. The trigger regulates the start of each breath. The limit controls inspiration to avoid excessive volume or pressure.



For the purposes of the Pediatric Fundamental Critical Care Support course, volume-preset ventilation is generally preferred over pressure-preset ventilation to assure appropriate tidal volume is maintained.

The cycling controls the termination of inspiration. Early positive pressure ventilators had no mechanism to detect patient effort; this sometimes resulted in dyssynchrony and poor tolerance. Newer ventilators can detect changes in pressure or flow as the patient inhales.

2. Volume and Pressure

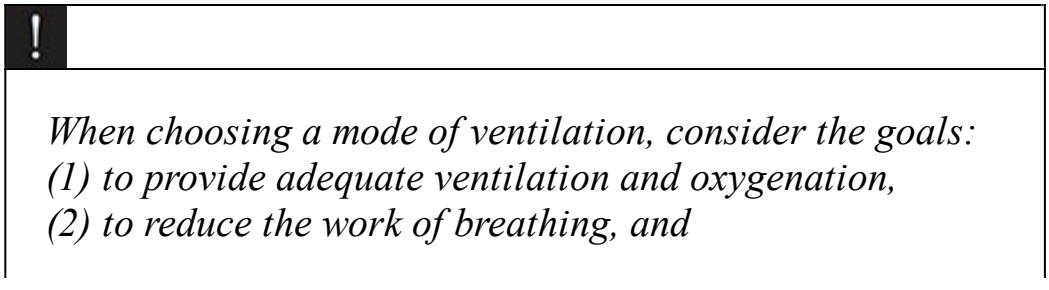
Common ventilator modes are limited by either volume or pressure. Targeting tidal volume (V_T) with volume-preset ventilation is generally preferred over pressure-preset ventilation. In the past, infants were ventilated primarily with pressure-preset modes due to limitations in flow delivery and imprecise V_T measurement in older ventilators. With newer machines, nearly all patients, including premature infants, can be managed using a volume-preset mode.

3. Time Cycling, Flow Cycling, and Other Variables

Ventilator modes are typically time-cycled; in other words, inspiration is terminated after a specified time. Flow cycling is used in specialized modes of ventilation, such as pressure support.

IV. MODES OF MECHANICAL VENTILATION

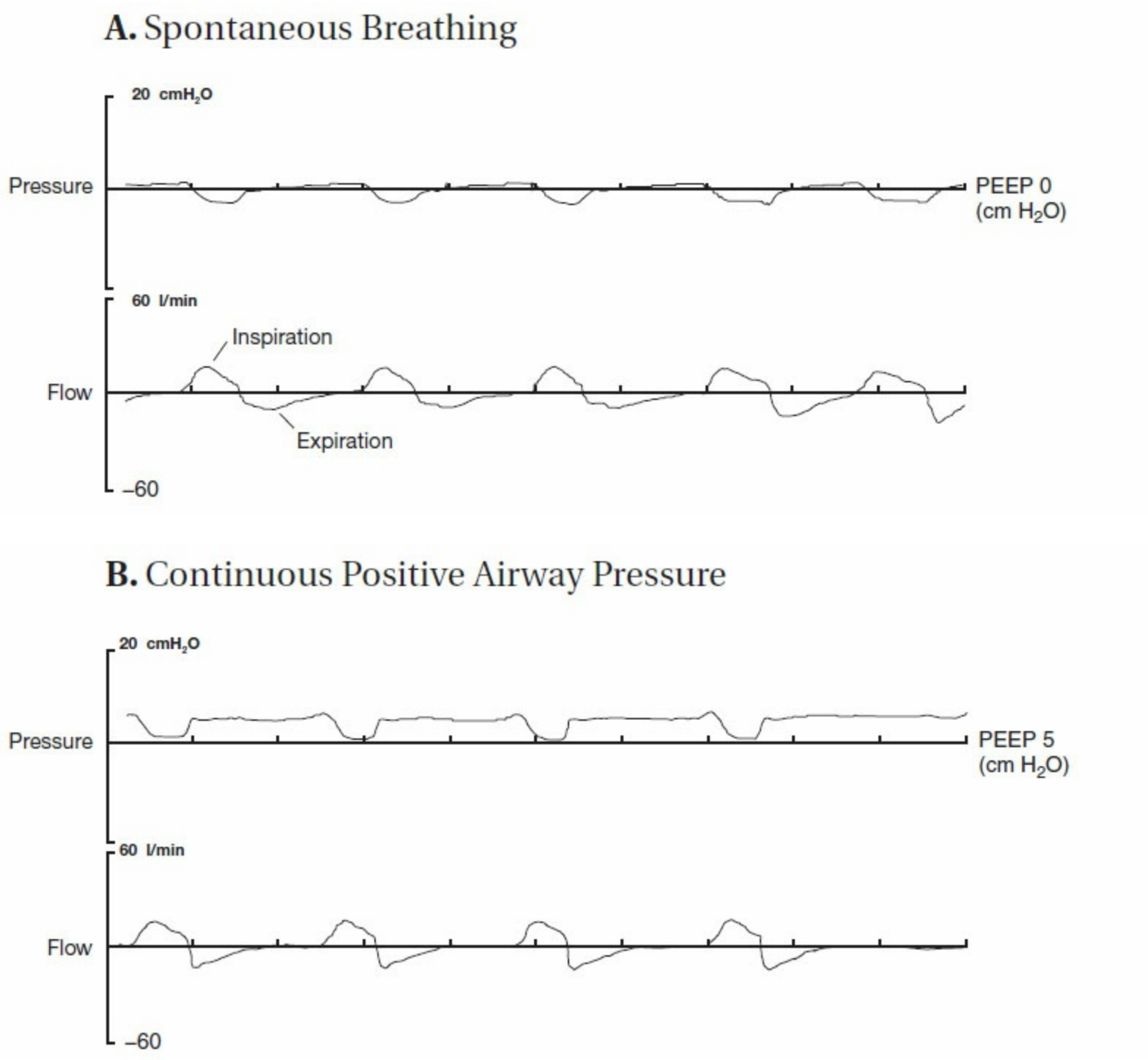
The choice of ventilator mode depends on how much of the function of gas exchange can be contributed by the patient and how much must be done by the machine. A trial of NIV may be considered in some circumstances. Frequently used ventilation modes include assist-control ventilation, synchronized intermittent mandatory ventilation, and pressure-support ventilation (**Figure 5-2**). Each has advantages and disadvantages, as summarized in **Table 5-3**.



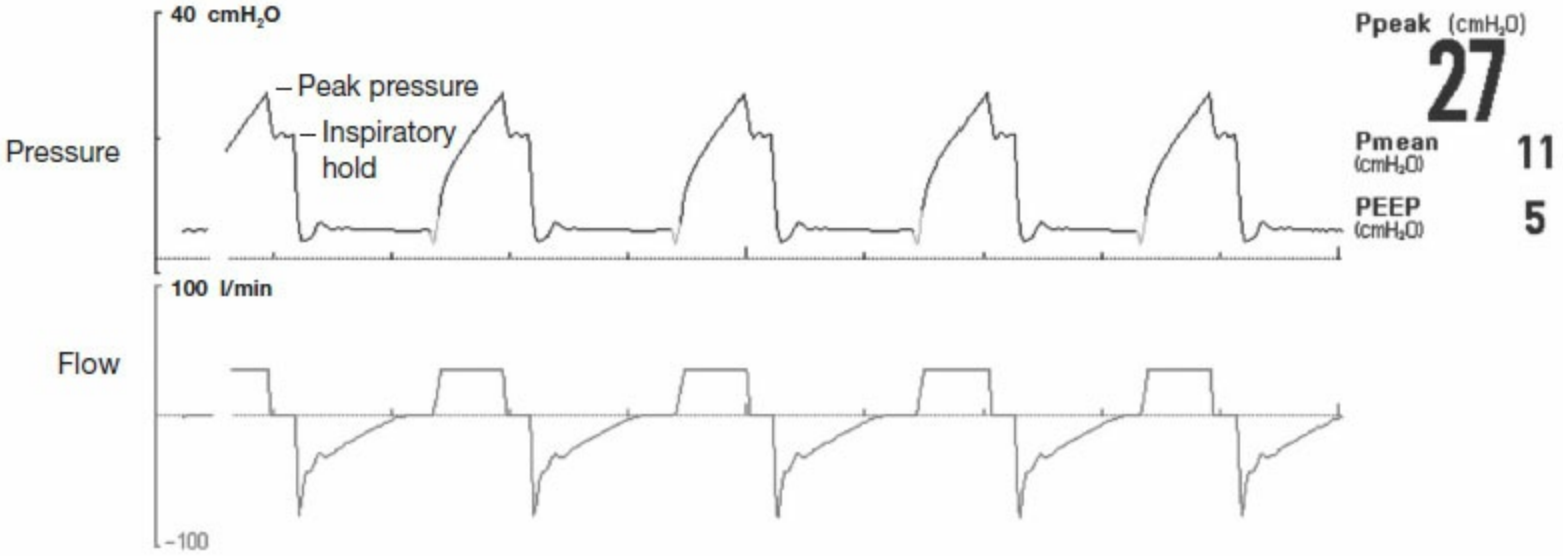
When choosing a mode of ventilation, consider the goals:
(1) to provide adequate ventilation and oxygenation,
(2) to reduce the work of breathing, and

(3) to ensure patient comfort and synchrony with the ventilator.

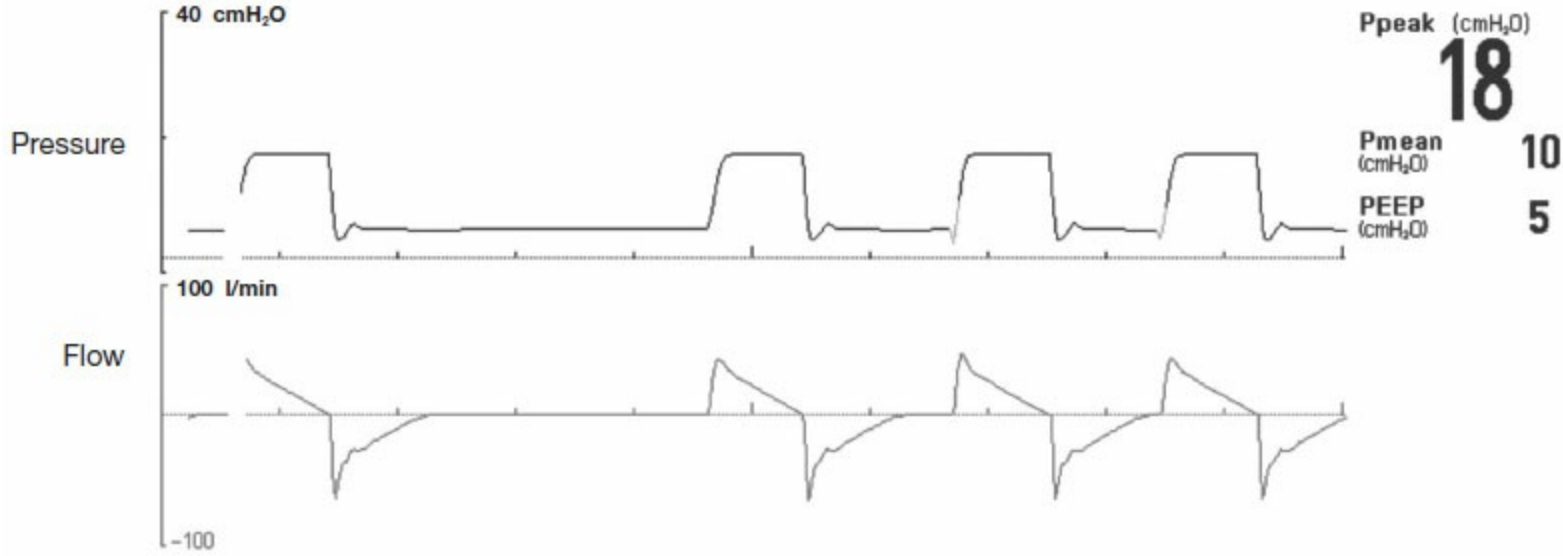
Figure 5-2. Airway Pressure and Flow Tracings



C. Assist-Control (AC) Volume Ventilation

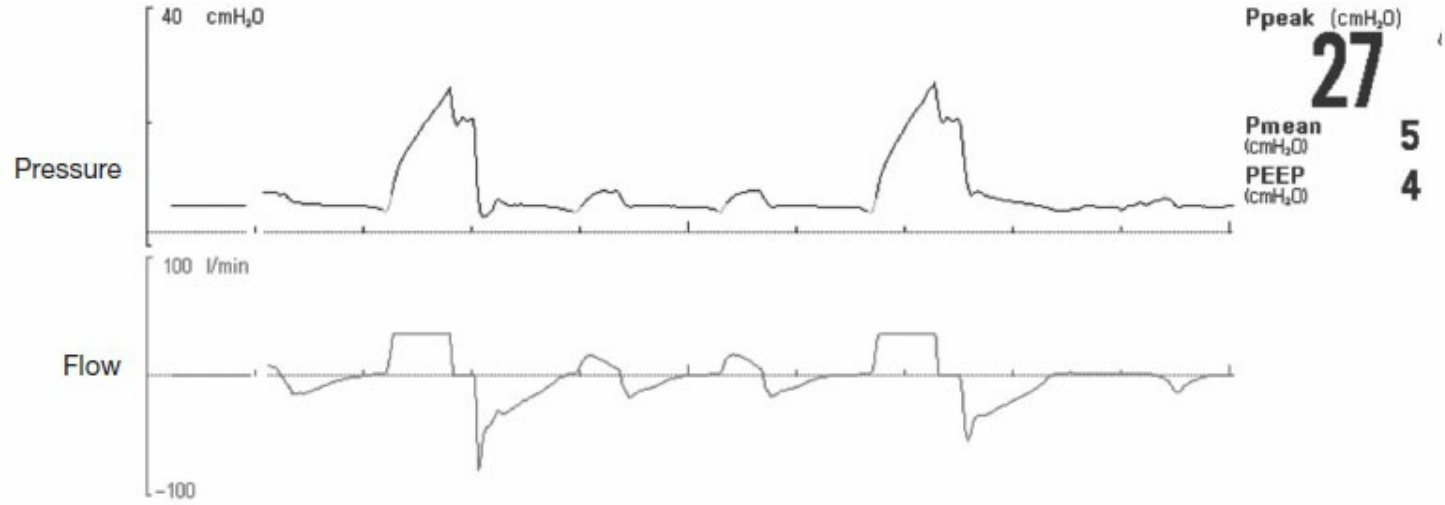


D. Assist-Control (AC) Pressure Ventilation

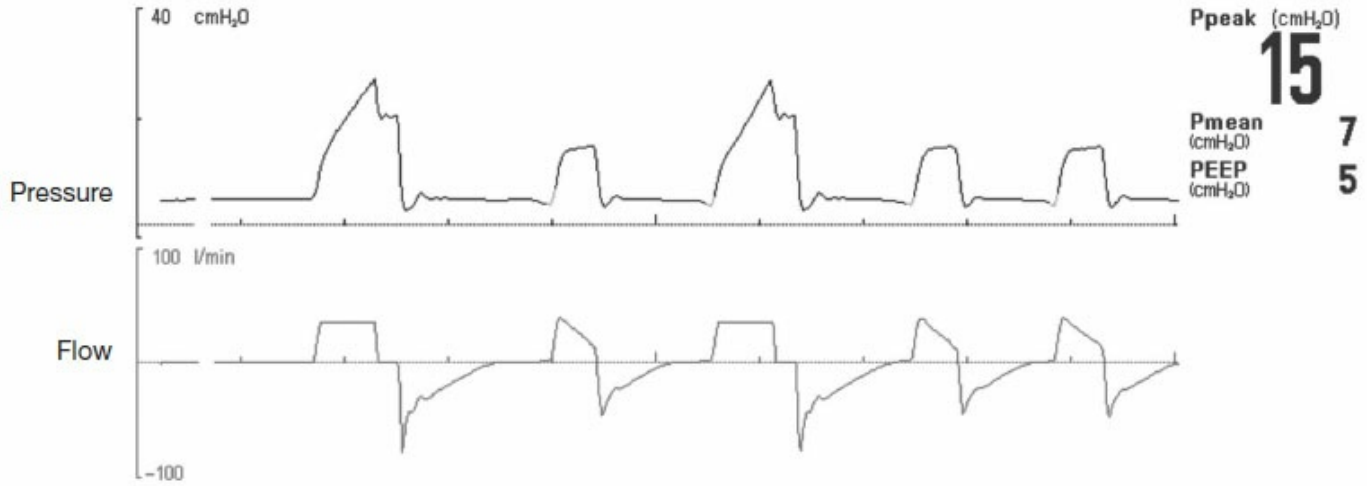


E. Pressure Support Ventilation

F. Synchronized Intermittent Mandatory Ventilation Without Pressure Support



G. Synchronized Intermittent Mandatory Ventilation With Pressure Support



Ppeak, peak inspiratory pressure; PEEP, positive end-expiratory pressure
Time is represented on the horizontal axis and pressure or flow on the vertical axis.

Tracings provided courtesy of Paul Ouellet, PhD(c), RRT, FCCM.

Table 5-3 POTENTIAL ADVANTAGES AND DISADVANTAGES OF SELECTED MODES OF MECHANICAL VENTILATION

Mode	Advantages	Disadvantages
AC ventilation	Patient can increase ventilatory support; reduced work of breathing compared to spontaneous breathing	Potential adverse hemodynamic effects; delivery of full breath with each patient effort may lead to inappropriate hyperventilation, breath stacking, and inadvertent positive end-expiratory pressure
AC volume ventilation	Guarantees delivery of set tidal volume (unless peak pressure limit is exceeded)	May lead to excessive inspiratory pressures
AC pressure ventilation	Allows limitation of peak inspiratory pressures	Potential hyper- or hypoventilation with lung resistance/compliance changes
Synchronized intermittent mandatory ventilation	Less interference with normal cardiovascular function	Increased WOB compared to AC ventilation; added patient-ventilator synchrony
Pressure support ventilation	Patient comfort; improved patient-ventilator interaction; decreased WOB	Apnea alarm is only backup; variable patient tolerance; only supports spontaneous breaths; patient must trigger ventilator for support

AC, assist-control; WOB, work of breathing.

A. Assist-Control Ventilation

Assist-control (AC) ventilation is controlled mechanical ventilation with a triggering mechanism that allows synchronization with the patient's respiratory effort. A preset volume or pressure is delivered at a preset minimum rate. A full ventilator breath is delivered with each spontaneous inspiratory effort; therefore, patients who are tachypneic due to their illness or agitation are likely to hyperventilate in this mode. AC ventilation is commonly used when mechanical ventilation is initiated in patients with acute respiratory failure when pulmonary compliance is not significantly abnormal.

With proper use of AC ventilatory support, the work of breathing may be significantly decreased. However, if the ventilator and the patient are not in synchrony or if the ventilator flow rates are not matched with patient demand, this mode of support may actually lead to an increase in the work of breathing.

Decreasing the work of breathing can be important for clinical situations in which there is inadequate cardiac output. Resting the diaphragm and respiratory muscles may allow redistribution of blood flow and improved delivery of oxygen to vital organ systems.

B. Synchronized Intermittent Mandatory Ventilation

Synchronized intermittent mandatory ventilation (SIMV) delivers either volume-preset or pressure-preset breaths at a preset rate. Most frequently, it is used with breaths in a volume-preset mode.

Unlike AC ventilation, no additional ventilator breaths are delivered above the preset rate. Spontaneous breathing above that rate is possible, but the V_T is limited to that which the patient can generate, unless pressure support is added. The use of synchronization allows for enhanced patient-ventilator interaction by delivering the preset machine breaths in conjunction with the patient's inspiratory effort. When no effort is sensed, the ventilator delivers the preset V_T at the set rate. SIMV is often combined with pressure-support ventilation (discussed later) to augment the V_T during spontaneous breathing. Pressure support should be set to deliver an appropriate spontaneous V_T and offset endotracheal tube (ETT) resistance, usually 5 to 10 cm H_2O.

One advantage of the SIMV mode is that it allows the patient to contribute to the minute ventilation requirement with spontaneous, negative-pressure breaths. The negative inspiratory pressure (relative to PEEP, not atmospheric pressure) leads to an increased venous return to the right side of the heart, which may improve cardiac output and cardiovascular function.

C. Pressure-Support Ventilation

Pressure-support ventilation (PSV) provides a preset level of inspiratory pressure with each spontaneous breath. All breaths are flow-cycled and pressure-limited. This inspiratory assistance is generally selected to overcome the increased work of breathing imposed by the disease process, the ETT, the ventilator inspiratory valves, and other mechanical aspects of ventilatory support. The pressure support augments each patient-generated, spontaneous breath. The patient controls the respiratory rate and exerts a strong influence on the duration of inspiration, inspiratory flow rate, and V_T. The delivered V_T is influenced by pulmonary compliance and resistance. Rapid changes in these parameters may alter the minute ventilation and respiratory work. PSV can also be coupled with SIMV, primarily as a means to diminish work of breathing during spontaneous breaths.

The amount of pressure support set during mechanical ventilation is titrated to the patient's exhaled spontaneous V_T. It should be set to achieve adequate gas exchange and optimize patient comfort. PSV only supports spontaneous breathing; when used alone, it will not provide ventilation during periods of apnea. Therefore, a backup, mandatory ventilation setting is required and is standard on many ventilators. Potential benefits of PSV include improved comfort and tolerance for awake, spontaneously breathing patients and reduced work of breathing due to enhanced patient-ventilator interaction. Typically, as pressure support is increased in patients with lung disease, the patient's work of breathing and respiratory rate decrease and V_T increases. Alternatively, patients recovering from severe lung disease who were initially placed on high levels of pressure support can be effectively weaned from mechanical ventilation by gradually decreasing the pressure support, thus requiring progressively more work from deconditioned respiratory muscles. The presence of a bronchopleural fistula or an ETT cuff leak may interfere with appropriate cycling in this mode due to the escaping gas.

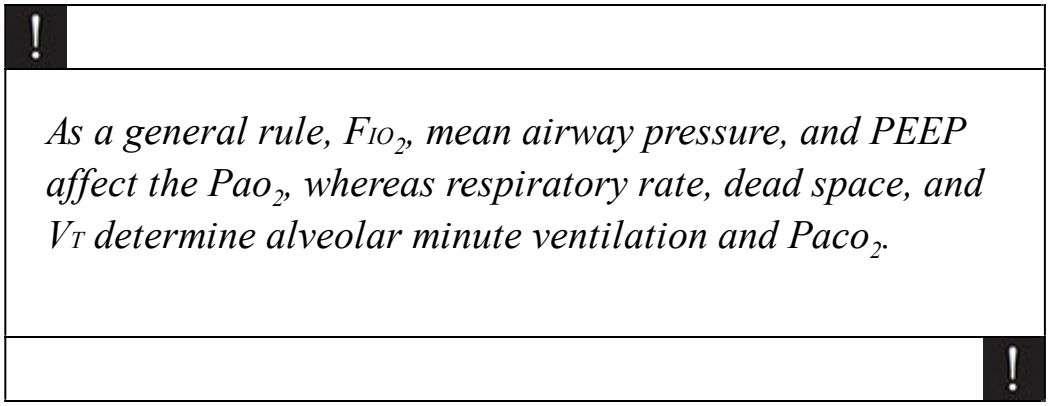
D. Other Modes of Mechanical Ventilation

Other modes of ventilation include airway pressure release ventilation and high-frequency oscillating ventilation. Need for these modes suggests a consultation with a pediatric critical care specialist is

needed.

V. INITIAL VENTILATOR SETTINGS

When initiating ventilatory support, an F_{IO_2} of 1.0 is generally used to maximize the oxygen available during the transition to positive pressure ventilation and to compensate for any periods of respiratory or hemodynamic instability. Tidal volumes are set between 6 and 8 mL/kg in patients with reasonably normal lung compliance. Lower volumes are considered when lung compliance is reduced (discussed later) or for the neonate (**Chapter 1**); higher volumes are generally avoided to decrease the risk of lung trauma. The choice of the initial respiratory rate in children is usually based on age and disease process and is subsequently titrated by measurement of pH and, to a lesser extent, $Paco_2$ in arterial blood. Guidelines for initiation of mechanical ventilation are listed in **Table 5-4**.



As a general rule, F_{IO_2}, mean airway pressure, and PEEP affect the Pao_2, whereas respiratory rate, dead space, and V_T determine alveolar minute ventilation and $Paco_2$.

Table 5-4 GUIDELINES FOR INITIATION OF MECHANICAL VENTILATION

1. Choose a ventilator mode with which you are familiar. The primary goals of ventilatory support are adequate oxygenation/ventilation, reduced work of breathing, synchrony between the patient and ventilator, and avoidance of high end-inspiration alveolar pressures (maintain plateau pressure <30 cm H_2O).
2. Use initial F_{IO_2} level of 1.0; thereafter it can be titrated downward to maintain an Spo_2 of 92% to 94%. In severe ARDS, Spo_2 ≥88% may be acceptable, lowering the therapeutic goal in favor of minimizing ventilator-associated lung injury. The exception to this recommendation is the patient with congenital heart disease, whose condition will be exacerbated by oxygen-driven decreases in pulmonary vascular resistance.
3. Set the initial V_T between 6 and 8 mL/kg and adjust according to blood gas analysis results. Patients with neuromuscular disease often require V_T levels of 10-12 mL/kg to satisfy air hunger. In some patients with ARDS, a V_T level of 4-6 mL/kg is recommended to avoid high inspiratory plateau pressures (>30 cm H_2O).
4. Choose a respiratory rate and minute ventilation appropriate for the particular clinical requirements. This rate is dependent upon age and disease process, generally 12 breaths/min (adolescents) to 24 breaths/min (neonates). Target near normal pH primarily and $Paco_2$ secondarily.
5. Set TI for each breath according to the patient's age and disease process. Neonates with normal lung function require TI of 0.35-0.6 second, whereas that for children older than 2 years is generally 0.85-1.0 second. In addition, the I:E ratio should be set with the patient's specific needs in mind. Start with a ratio of 1:2 in patients with healthy lungs. Patients with respiratory illnesses marked by difficulty with oxygenation (ARDS) may require a TI >1 and I:E ratio >1:1, whereas patients with obstruction to outflow (as in asthma and bronchopulmonary dysplasia) will require ample expiratory time and I:E ratios of 1:3.5 to 1:4 to avoid auto-PEEP (see below). Parameters for inspiration and exhalation depend somewhat on rate. Critical care consultation is advised for patients requiring inverted I:E ratio.

6. Use PEEP to achieve and maintain optimal alveolar recruitment. In diffuse lung injury, PEEP will improve oxygenation and reduce the F_{IO_2} (**Table 5-5**). If volume is held constant, PEEP increases mean airway pressure, but it may also increase peak inspiratory airway pressure, depending on ventilator mode; this may result in potentially undesirable consequences in ARDS. Set PEEP to 5 cm H_2O when initiating mechanical ventilation in children with healthy lungs. PEEP levels >15 cm H_2O are rarely necessary.
7. Set the trigger sensitivity to allow minimal patient effort to initiate inspiration. Beware of autocycling if the trigger setting is too sensitive.
8. Consider sedation, analgesia, and/or neuromuscular blockade when poor oxygenation, inadequate ventilation, or excessively high peak inspiratory pressures are thought to be related to patient intolerance of ventilator settings and cannot be corrected by ventilator adjustments.
9. Call a pediatric critical care consultant or other specialist in respiratory care for assistance.

Spo_2, peripheral oxyhemoglobin saturation; ARDS, acute respiratory distress syndrome; V_T, tidal volume; TI, inspiratory time; I:E ratio, ratio of inspiratory to expiratory time; PEEP, positive end-expiratory pressure

Table 5-5 USE OF POSITIVE END-EXPIRATORY PRESSURE IN ACUTE LUNG INJURY/ACUTE RESPIRATORY DISTRESS SYNDROME

Initiation of PEEP in Acute Respiratory Distress Syndrome

- PEEP at 5 cm H_2O and titration in increments of 2-3 cm H_2O
- Full recruitment effect may not be apparent for several hours
- Monitoring of blood pressure, heart rate, and Pao_2 during PEEP titration and at intervals while the patient is receiving PEEP therapy
- Optimal PEEP settings are typically 8-15 cm H_2O

Adverse Effects of PEEP

- Barotrauma
- Decreased venous return, decreased preload, decreased cardiac output, and hypotension
- Increase in $Paco_2$ (increase in dead space)
- Worsening oxygenation (especially with asymmetric acute lung injury) as healthy lung is overdistended

PEEP, positive end-expiratory pressure.

VI. CONTINUING CARE DURING MECHANICAL VENTILATION

After mechanical ventilation has been initiated, the parameters listed below should be assessed frequently to guide changes in the ventilator settings. Care should be taken when any change is made because adjusting one parameter is likely to affect the others. This interdependency may cause a change that is beneficial in one respect to be harmful in another. In complex situations, critical care consultation should be obtained.

A. Fraction of Inspired Oxygen

Inspired oxygen may be harmful to the lung parenchyma after prolonged exposure due to the production of toxic oxygen-derived radicals. Although the precise threshold for concern is not known, it is desirable to reduce the F_{IO_2} to 0.5 (50% oxygen) or below as soon as possible, ideally within the first 24 hours. Except in the premature neonate, to whom oxygen toxicity may be equally damaging, hypoxemia is usually considered a greater risk to the patient than high F_{IO_2} levels.

The primary determinants of oxygenation during mechanical ventilation are F_{IO_2} and mean airway

pressure. In the patient with acute lung injury or acute respiratory distress syndrome, PEEP becomes an additional independent determinant. V_T, the relationship of inspiratory time to expiratory time (I:E ratio), PEEP, and several other factors beyond the scope of this chapter, all interact to affect the mean airway pressure. The interrelationships of those factors, as already noted, must be considered when formulating the plan for mechanical ventilation.

B. Humidification

Gases delivered by mechanical ventilators are typically dry, and the upper airways are bypassed by artificial airways. These circumstances result in loss of heat and moisture from the respiratory tract. Gases are routinely heated and humidified during mechanical ventilation to prevent mucosal damage and minimize inspissation of secretions. Available systems include passive humidifiers (artificial nose) or active, microprocessor-controlled heat and humidifying systems (heated humidifiers). Passive humidifiers are contraindicated in the presence of copious secretions, minute ventilation >12 L/min, or blood in the airway.

C. Inspiratory Pressure

During positive pressure ventilation, airway pressure rises progressively to a peak inspiratory pressure (Ppeak). Ppeak may also be abbreviated as *PIP* or referred to as *peak pressure* or *peak airway pressure*. Ppeak is the sum of 2 pressures: the pressure required to overcome airway resistance and that required to overcome the elastic properties of the lung and chest wall. If a pause is set at the end of inspiration (zero gas flow with the airway closed), the pressure drops slightly below the Ppeak to the inspiratory plateau pressure (Pplat) [**Figure 5-3**]. Pplat reflects the pressure required to overcome elastic recoil and is the best estimate of peak alveolar pressure, which is an important indicator of alveolar distension. Accurate measurement of Ppeak requires the absence of any patient effort during inspiration or exhalation.

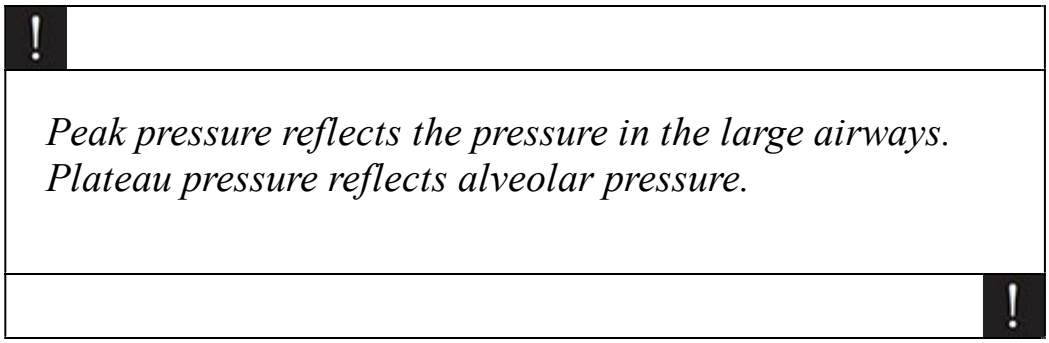
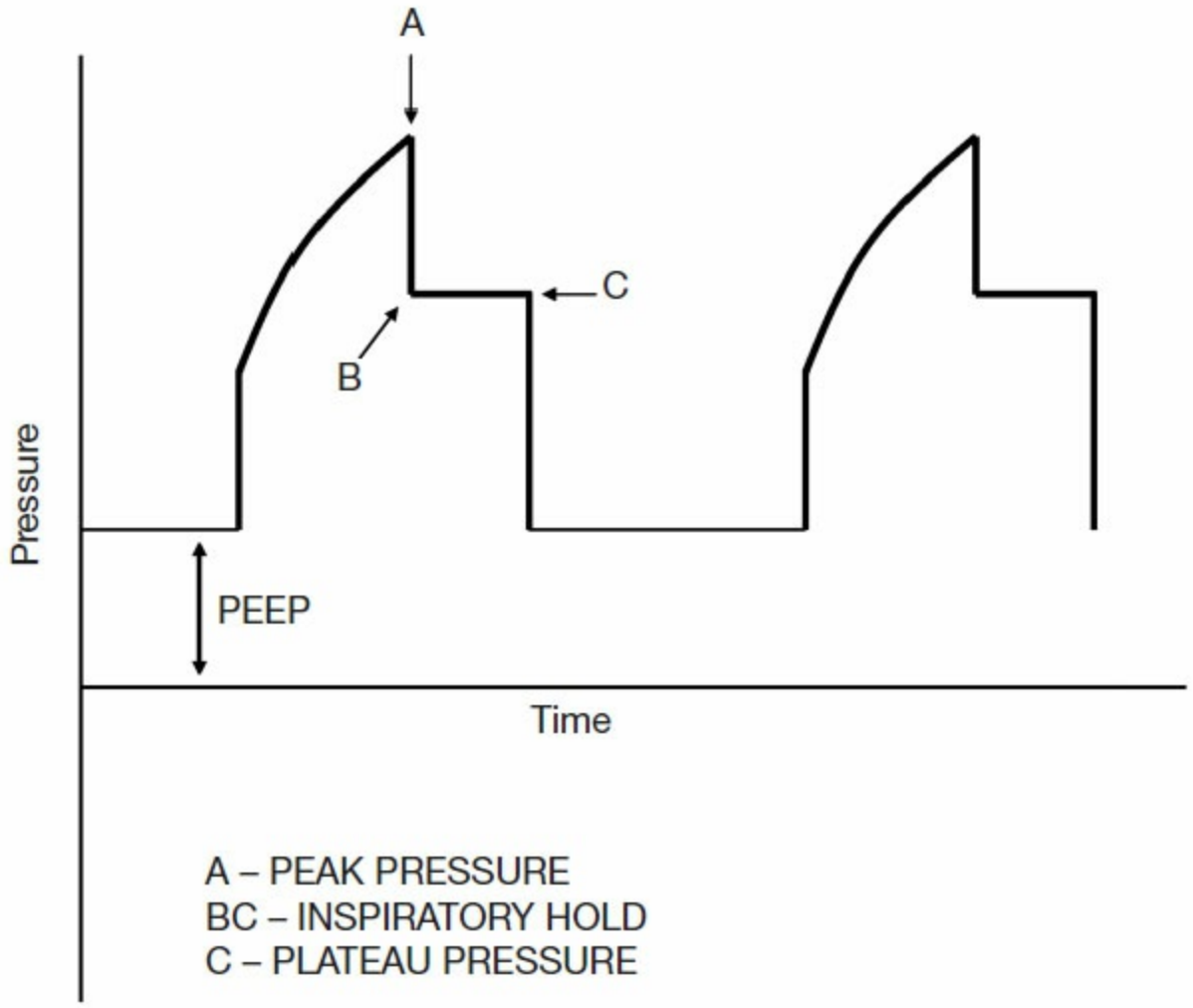
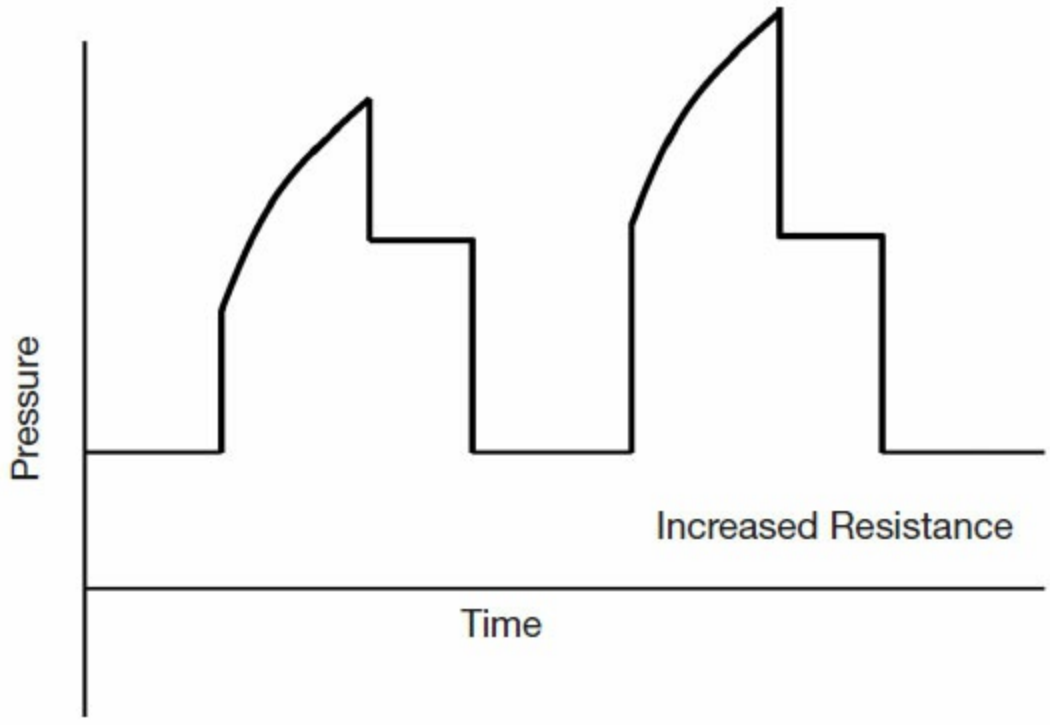
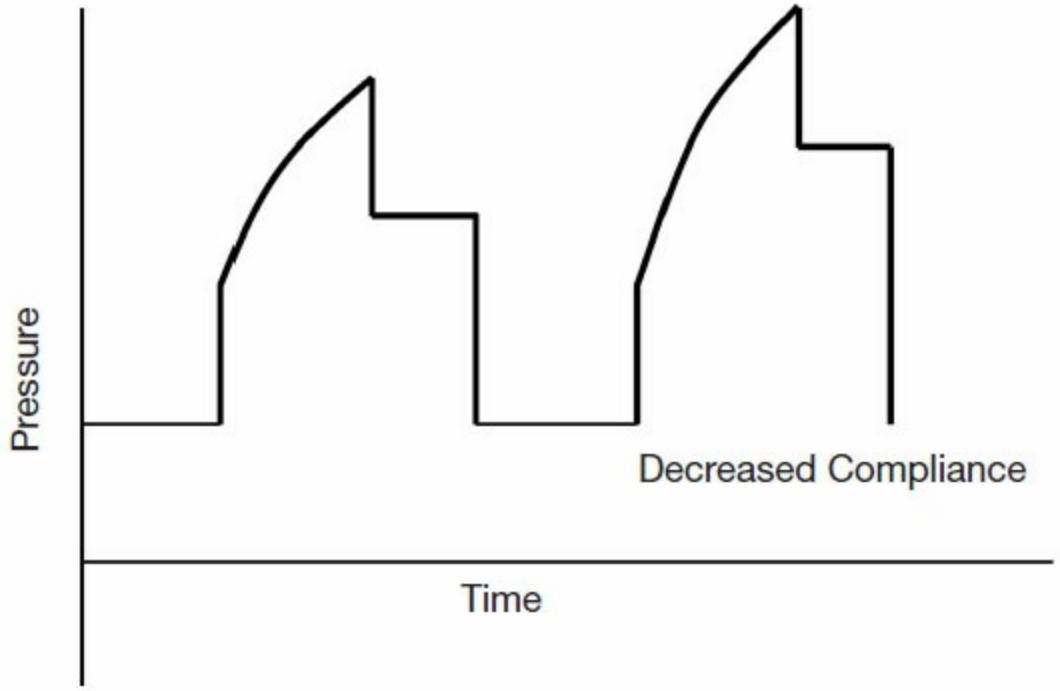


Figure 5-3. Relationship of Peak Inspiratory Pressure and Inspiratory Plateau Pressure



A – PEAK PRESSURE
BC – INSPIRATORY HOLD
C – PLATEAU PRESSURE





Potential adverse effects from high inspiratory pressure include barotrauma, volutrauma, and reduced cardiac output. Barotrauma (pneumothorax, pneumomediastinum) and volutrauma (lung parenchymal injury due to overinflation), although linked to high Ppeak, correlate best with Pplat, a better measurement of alveolar pressure. An example of the relationship of Ppeak and Pplat to alveolar distension is demonstrated by the effect of ETT size on Ppeak and Pplat. As the internal diameter of the ETT is decreased in a patient receiving volume ventilation, the same V_T will result in higher Ppeak, yet Pplat and alveolar distension remain unchanged as the pressure is dissipated across the ETT. The same V_T, regardless of the type of breath, produces the same alveolar distension at end inspiration. Ideally, Pplat should be <30 cm H_2O.

Elevated Pplat may be reduced by decreasing PEEP, which may also decrease oxygenation, or by decreasing V_T, which may lead to a reduction in minute ventilation. Permissive hypercapnia and a controlled reduction in pH may be accepted when high Pplat must be reduced. Permissive hypercapnia should not be used in patients with elevated intracranial pressure because $Paco_2$ >35 to 40 mm Hg (>4.7-5.3 kPa) may increase cerebral blood flow and cerebral blood volume and further elevate the intracranial pressure.

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Ideally, Pplat should be <30 cm H_2O. An elevated plateau pressure can be decreased by decreasing PEEP or decreasing the V_T.

!

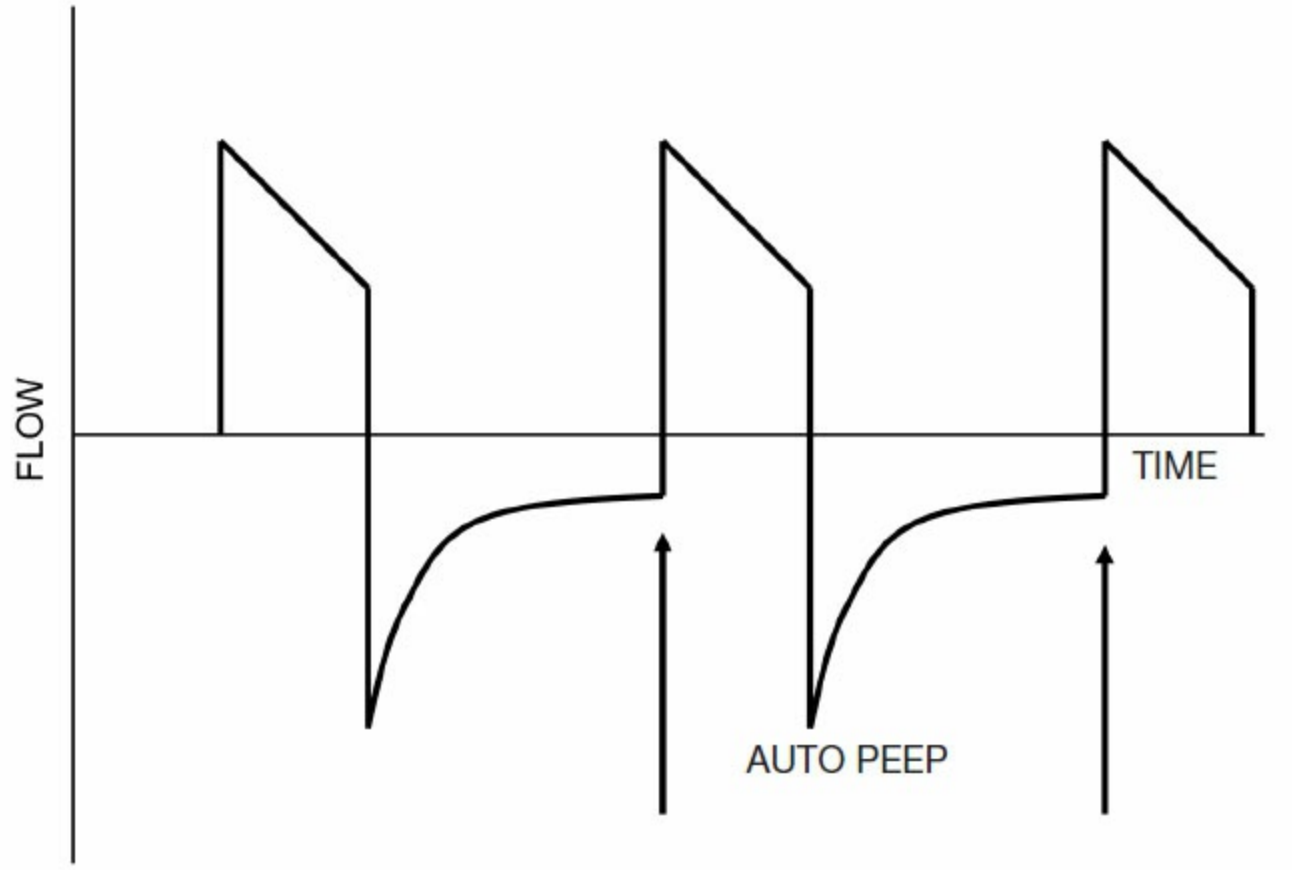
D. Relationship of Inspiratory Time to Expiratory Time and Auto-PEEP

The total respiratory cycle time is calculated by dividing 60 seconds by the respiratory rate. The times for inhalation (inspiratory time) and exhalation (expiratory time) occur within the total respiratory cycle time. When considered as relative portions of the respiratory cycle, they are termed the *I:E ratio*. During spontaneous breathing, the normal I:E ratio is ~1:2, indicating that the exhalation time is about twice as long as inhalation time in normal patients. In patients with chronic lung disease and other conditions associated with obstruction to expiratory flow, such as asthma, the exhalation time becomes prolonged, and the I:E ratio changes (eg, 1:2.5 or 1:3). Such changes reflect the pathophysiology of the lung disease and directly influence techniques used during mechanical ventilation.

The inspiratory time during volume breaths (AC or SIMV modes) is often determined by the V_T and inspiratory gas flow rate (flow × inspiratory time = V_T). A greater V_T takes longer to deliver if the flow rate remains constant, and the same V_T takes longer to deliver if the flow rate is reduced. In both cases, the inspiratory time is longer. However, given that the respiratory rate is constant, the total cycle time remains the same, and thus the time available for exhalation (expiratory time) must be shortened to fit into the cycle time. In this situation, the inspiratory time is actively set by adjusting V_T and the inspiratory flow rate or by the use of inspiratory pauses on the ventilator. The expiratory time, however, is passively determined (ie, what is left over in the cycle time before the next inspiratory cycle of the mechanical ventilator or the spontaneous breath) and varies inversely with the respiratory rate set on the ventilator.

If the expiratory time is too short to allow full exhalation, the next lung inflation is superimposed upon the residual gas in the lung and breath stacking occurs. This process will result in hyperinflation of the lung and the development of PEEP that exceeds the level of PEEP preset on the ventilator. This increase in end-expiratory pressure is *auto-PEEP*, or *intrinsic*, *inadvertent*, or *occult PEEP*. Auto-PEEP can be quantified by using manual methods or through electronic programs available in some ventilators. It is most easily diagnosed qualitatively by the flow-over-time graphic waveform tracing available on most mechanical ventilators (**Figure 5-4**). The potentially harmful physiologic effects of auto-PEEP on Ppeak, Pplat, and mean airway pressure and on lung and cardiovascular function are the same as those of preset PEEP (**Table 5-5**). Auto-PEEP may also increase the work of breathing because the difference between the set PEEP and auto-PEEP must be overcome on inspiration to allow for fresh gas flow.

Figure 5-4. Flow-Versus-Time Waveform Demonstrating Auto-PEEP



PEEP, positive end-expiratory pressure

Reduction in auto-PEEP can be accomplished by:

- Shortening the inspiratory time. This extends the expiratory time to allow full exhalation of the V_T; however, it may cause alveoli with longer time constants to fill incompletely.
- Decreasing the respiratory rate. This is the most effective way to decrease auto-PEEP.
- Decreasing V_T. Increasing the inspiratory flow rate (volume breaths) is typically less effective unless flow rates are set inappropriately low. The influence of a decreased respiratory rate and V_T on the $Paco_2$, pH, and minute ventilation must be considered.

E. Minute Ventilation

The primary determinant of CO_2 exchange during mechanical ventilation is alveolar minute ventilation (V_A), calculated as:

$$V_A = (V_T - V_{DS}) \times f$$

where V_{DS} is dead space and f is respiratory rate. The V_T, the respiratory rate, and their interrelationships with other ventilatory parameters have already been noted. Physiologic V_{DS} represents, in general, lung units that are relatively well ventilated but underperfused. The physiologic effect of increased V_{DS} is hypercapnia. Increased physiologic V_{DS} may result from the pathologic process in the lung, high airway pressures associated with mechanical ventilation, low intravascular volume, or low cardiac output. If hypercapnia persists during mechanical ventilation

despite use of appropriate V_T and rates, consultation with a pediatric intensivist should be sought. It may be necessary to tolerate a higher $Paco_2$ to avoid high airway pressure and auto-PEEP. This permissive hypercapnia technique should be initiated only with the support of an appropriate pediatric critical care consultant.

Adequate ventilation is assessed by consideration of both $Paco_2$ and pH. Hyperventilation resulting in a low $Paco_2$ may be an appropriate short-term compensatory goal during metabolic acidosis until the primary problem can be corrected. Similarly, a patient with chronic hypercapnia and increased $Paco_2$ at baseline, who maintains a near-normal pH by renal compensation (retention of bicarbonate), should receive only enough minute ventilation to maintain a normal pH. Driving $Paco_2$ to normal levels in the patient with chronic CO_2 retention will lead to severe alkalemia and loss of retained bicarbonate and may blunt the patient's respiratory drive.

VII. SEDATION, ANALGESIA, AND NEUROMUSCULAR BLOCKADE

Endotracheal intubation and mechanical ventilation can be painful, frightening, and anxiety-provoking. To improve patient comfort, relieve anxiety, and lessen the work of breathing, anxiolytics, sedatives, analgesics, and neuromuscular blocking agents are frequently administered. Neuromuscular blocking agents should only be used when absolutely necessary. Adequate sedation and analgesia must be assured use before neuromuscular blockade. Guidelines for the use of these agents are outlined in **Chapter 20** and **Appendix 7**.

VIII. VENTILATORY GUIDELINES FOR SPECIFIC CLINICAL SITUATIONS

A. Acute Lung Injury/Acute Respiratory Distress Syndrome

Case Study

A 16-year-old boy who underwent splenectomy 2 months ago presents to the emergency department with a 3-day history of fevers (38.8°C [101.8°F]), shaking, chills, and increasing shortness of breath. Oxygen saturations are 86% while breathing room air and increase to 90% on 100% non-rebreather face mask. On exam, his respiratory rate is 24 breaths/min, and coarse crackles can be heard throughout both lungs. All extremities are pale and cool, with a capillary refill of approximately 4 seconds. He is drowsy and disoriented. Analysis of an arterial blood gas drawn while on the non-rebreather mask shows: pH 7.26; $Paco_2$, 54 mm Hg (7.1 kPa); Pao_2, 62 mm Hg (8.1 kPa); base excess, -5 mEq/L; oxygen saturation, 88%. The chest radiograph shows bilateral patchy infiltrates.

Detection

– What is the most likely diagnosis in this patient?

– What is the significance of his history of splenectomy?

Intervention

– What is the best next intervention in the management of this patient?

– Is this patient a good candidate for NIV? Why or why not?

Reassessment

– Is the current oxygenation and ventilation strategy effective?

– What assessments need to be done?

– What changes would you make at this time?

Effective Communication

– When changes are made or the patient's status changes, how will the information be communicated and to whom?

– Where is the best place to manage this patient?

Teamwork

– Who is involved in the patient's care?

– How will this patient's treatment be implemented?

Compromised or inadequate oxygenation is the hallmark of acute respiratory distress syndrome (ARDS), thus its alternative name, *acute hypoxemic respiratory failure*. Infection or inflammation in the lungs triggers the release of chemical mediators that disturb normal lung function, cause capillary leak, and increase intrapulmonary shunting, thereby worsening ventilation-perfusion mismatch. Poor lung compliance and increased airway resistance require the use of high pressures during mechanical ventilation to restore adequate gas exchange, and the challenge lies in how best to support respiration while causing the least amount of trauma to the lungs. The landmark ARDS Network study, published in 2000, showed that the use of low V_T (6 mL/kg) compared to standard V_T (12 mL/kg) resulted in decreased mortality rates and more ventilator-free days for adults with acute lung injury and ARDS. A similar strategy has been widely adopted in pediatric ICUs throughout the world. The latest consensus on pediatric ARDS recommends V_T as low as 3-6 mL/kg for patients with poor respiratory system compliance and 5-8 mL/kg for patients with preserved compliance. Strategies borrowed from the neonatal experience with hyaline membrane disease—such as high-frequency oscillatory ventilation, surfactant instillation, and inhaled nitric oxide—have also been employed in pediatric patients with ARDS, but none has been shown to improve outcomes in large prospective randomized clinical trials.

Although the strategy for mechanical ventilation in patients with ARDS focuses on optimizing

oxygenation, care must be taken to avoid breath stacking and inadvertent auto-PEEP that may result from the use of prolonged inspiratory time and inverse I:E ratios.

PEEP helps to recruit and maintain open lung segments to optimize gas exchange.

B. Obstructive Airway Disease

Case Study

An 11-year-old, 50-kg girl with a history of mild intermittent asthma presents to the emergency department complaining of chest pain and shortness of breath that has worsened over the past 2 days. Her respiratory rate is 36 breaths/min, and oxygen saturations are 92% on room air. Examination reveals mild suprasternal retractions, and only minimal air entry is appreciated on auscultation. Continuous inhaled bronchodilators and methylprednisolone are given without improvement, and the patient is transferred to the pediatric ICU, where a terbutaline infusion is started. Her heart rate increases to 180 beats/min with S-T wave elevations.

Soon after arrival, the patient becomes drowsy and disoriented. As she is being prepared for intubation, an arterial blood gas analysis shows the following values: pH 7.14; $Paco_2$, 80 mm Hg (10.7 kPa); Pao_2, 76 mm Hg (10.1 kPa); base excess, -8 mEq/L; and oxygen saturation, 88%. She is emergently intubated, and positive pressure ventilation is initiated in SIMV–volume-control mode with the following settings: respiratory rate, 12 breaths/min; V_T, 350 mL; F_{IO_2}, 1.0; PEEP, 5 cm H_2O; inspiratory time, 1 second; I:E ratio, 1:4.

Detection

– What are the potential risks and benefits of endotracheal intubation and mechanical ventilation in this patient with severe status asthmaticus?

Intervention

– What are important considerations when setting the ventilator parameters in this patient?

– What is the indication for PEEP in a patient with obstructive lung disease whose lungs are already hyperinflated?

Reassessment

– What should be monitored in a patient with severe status asthmaticus?

– What changes are necessary if auto-PEEP is detected?

Effective Communication

– What clinical changes require communication?

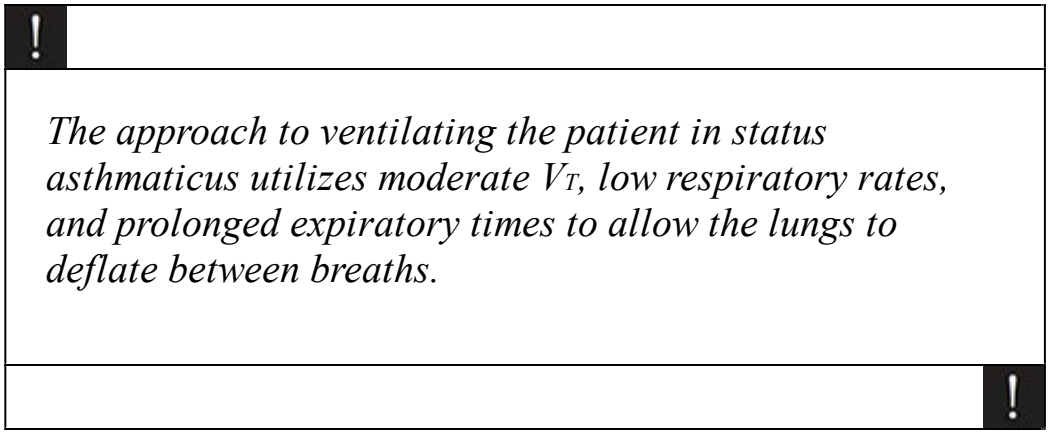
– What level of care does this patient need?

Teamwork

– What team members should be involved?

– How will the treatment plan be implemented?

Intubation and mechanical ventilation of the patient with status asthmaticus should be an intervention of last resort, utilized only after all other measures have been exhausted. Laryngoscopy can trigger profound bronchospasm and should be performed by the most capable and experienced healthcare provider available. The lungs of patients with respiratory failure due to asthma are generally hyperinflated, and the risk of pneumothorax and pneumomediastinum is high.


!
The approach to ventilating the patient in status asthmaticus utilizes moderate V_T, low respiratory rates, and prolonged expiratory times to allow the lungs to deflate between breaths.
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The use of PEEP in these patients is controversial and stylistic. Large randomized controlled studies comparing the use of physiologic PEEP to no PEEP in the management of children receiving mechanical ventilation for severe asthma have not been conducted. When the patient with asthma is intubated for hypercapnic respiratory failure, it is likely due to air trapping rather than hypoventilation, and setting a high respiratory rate on the ventilator to blow off CO_2 will not result in the desired outcome. Bronchospasm and small airway obstruction should be treated aggressively with parenteral or inhaled bronchodilators and corticosteroids to relieve the obstruction and reverse the acute process. Patients intubated for status asthmaticus should be extubated as soon as the bronchospasm is under control and they meet clinical criteria for extubation.

Published cases have reported pediatric patients who have developed cerebral edema in the setting of profound respiratory acidosis (with $Paco_2$ >100 mm Hg [13.3 kPa]) associated with status asthmaticus. Patients requiring intubation for hypercapnic respiratory failure should be monitored closely for signs of increased intracranial pressure.

C. Asymmetric Lung Disease

Asymmetric lung disease or injury that occurs after aspiration, contusion, localized pneumonia, or hemorrhage may cause abnormal distribution of ventilation and gas exchange during mechanical ventilation. Because the conditioned gas from the ventilator follows the path of least resistance along the bronchi, the V_T is distributed primarily to the less-affected (more compliant) lung and may overexpand it. Overdistension of the less-affected lung and poor expansion of the diseased or injured lung worsen ventilation-perfusion relationships in both lungs, and hypoxemia and hypercapnia may occur, persist, or worsen. Standard settings and principles of ventilatory support should be initiated. If this attempt is unsuccessful, expert consultation should be obtained regarding further patient management. Positioning the patient so that the healthier, better ventilated lung is in the gravitationally dependent position (sick side up) is a simple strategy for improving ventilation-perfusion matching and should result in improved oxygenation. Other techniques, such as differential lung ventilation, may be required.

D. Heart Disease

Case Study

A 3-month-old male infant with trisomy 21 (Down syndrome) and a complete atrioventricular canal defect is brought to his cardiologist 3 weeks after digoxin and furosemide have been initiated. His mother reports he still requires 45 minutes to ingest 30 mL of formula and then is too exhausted to take any more. He has gained only 150 g since birth and has fallen off his growth curve. On exam, the infant is breathing at a rate of 72 breaths/min with deep suprasternal retractions. His liver edge is firm and palpable 4 cm below the right costal margin. Extremities are pale and cool, and pulses are diminished.

Detection

– What respiratory signs or symptoms are detected?

– What is the likely cause of this patient's respiratory distress?

Intervention

– Would this patient be a good candidate for NIV? Why or why not?

– If the patient is placed on conventional ventilation, what settings would be appropriate?

– What other interventions are appropriate?

Reassessment

– What additional assessments must be completed in this patient?

– What is the best range for the oxygen saturation?

– How would the initiation of positive pressure ventilation impact this infant's circulatory status?

Effective Communication

– Where is the best place to manage this patient?

– If the patient's clinical status deteriorates, who needs to be contacted?

Teamwork

– Who should implement the treatment strategy?

– How should the responsibilities be divided?

The primary goal of ventilatory support in patients with myocardial ischemia or heart failure is to decrease the work of breathing and thereby ensure adequate oxygen delivery to the myocardium. Decreasing the work of breathing reduces consumption of oxygen by respiratory muscles and increases the amount of oxygen available to the heart and end organs. Patients with cardiogenic pulmonary edema may also benefit from the increased thoracic pressure with positive pressure ventilation, which decreases biventricular filling (lowering pulmonary capillary pressure) and unloads the left ventricle (increasing stroke volume).

High mean airway pressure during mechanical ventilation may adversely affect cardiac performance by increasing right ventricular afterload and reducing venous return. Therefore, the parameters that affect this pressure should be monitored and adjusted to maintain adequate cardiac function.

E. Neuromuscular Disease

Case Study

A 9-year-old boy is admitted to the pediatric ICU for observation after falling down a flight of stairs. His parents report that his knees "just gave out from under him." A review of systems is significant only for a bout of "walking pneumonia" 2 weeks ago, from which he recovered uneventfully. On examination, tenderness in both lower extremities, absent patellar reflexes, and decreased muscle tone are noted. On the night of admission, he develops difficulty swallowing, followed by respiratory distress requiring emergent intubation and positive pressure ventilation.

Detection

– What unique considerations must be addressed when ventilating the child with neuromuscular disease?

Intervention

– Would this patient be a good candidate for NIV? Why or why not?

– If the patient is placed on conventional ventilation, what settings would be appropriate?

Reassessment

– What additional assessments must be completed?

– What is the best target range for the pH?

Effective Communication

– Where is the best place to manage this patient?

– If the patient's clinical status deteriorates, who needs to be contacted?

Teamwork

– Who should implement the treatment strategy?

– How should the responsibilities be divided?

Patients with peripheral neuromuscular disease typically have an intact respiratory drive and normal lungs. They may require a higher V_T to avoid the sensation of dyspnea. Adjustments may need to be made in other ventilatory parameters to ensure a normal arterial pH. Whenever possible, supported spontaneous breathing with adequate pressure support should be encouraged to prevent further deterioration in respiratory muscle function. Resting settings with full ventilatory rate can be considered at night to promote rest.

IX. MONITORING MECHANICAL VENTILATORY SUPPORT

Patients who receive mechanical ventilatory support require continuous monitoring to assess the effects of treatment (**Table 5-6**). Arterial blood gas measurement provides valuable information about the adequacy of oxygenation, ventilation, and acid-base balance (**Appendix 3**). This information is mandatory during initiation of ventilator support, periods of patient instability, and weaning from mechanical ventilation. Chest radiographs can follow the evolution and resolution of disease processes, as well as verify ETT position.

Table 5-6	RECOMMENDATIONS FOR MONITORING MECHANICAL VENTILATORY SUPPORT

1. Obtain a chest radiograph after intubation and intermittently as needed to evaluate placement of the endotracheal tube and deterioration in status.
2. Obtain arterial blood gas measurements after initiation of mechanical ventilation and then intermittently based upon patient status.
3. Measure vital signs intermittently; frequently observe the patient directly (including evaluation of patient-ventilator interaction).
4. Measure inspiratory plateau pressure as clinically appropriate.
5. Use continuous pulse oximetry to measure oxygenation.

6. Use ventilator alarms to monitor key physiologic and ventilator parameters.

Ventilators are equipped with sophisticated alarms and monitors to assist with patient management and the detection of adverse events. When initiating ventilator support, the alarm parameters for low and high minute ventilation, high inspiratory pressures, and low exhaled volumes and pressures must be set. In addition, many ventilators allow for the measurement of auto-PEEP.

The low-pressure alarm is intended to alert the clinician to a leak in the circuit or ventilator disconnection. A high-pressure alarm warns that the set maximum Ppeak has been exceeded; this alarm is usually set 5 to 10 cm H_2O above the patient's baseline Ppeak. For a patient receiving volume ventilation, a high-pressure alarm may indicate the presence of a mucous plug, a sudden change in airway or lung compliance due to an accumulation of secretions, pneumothorax, or a change in ETT position. The patient may not receive the entire set V_T, because inspiration ends when the pressure limit is exceeded. For this reason, the frequent sounding of ventilator alarms should not be attributed to machine malfunction and should never be ignored or disabled.

X. HYPOTENSION WITH INITIATION OF MECHANICAL VENTILATION

A. Tension Pneumothorax

When hypotension occurs immediately after initiation of mechanical ventilation, a tension pneumothorax is possible. Diagnosis of this condition is based on a physical examination that finds decreased or absent breath sounds and tympanies to percussion on the side of the pneumothorax. Changes in vital signs may include decreased oxygen saturation, tachycardia, and an increased respiratory rate if the patient is breathing spontaneously. Tracheal deviation away from the side of the pneumothorax may be observed, although such a finding is uncommon after placement of an ETT. Ventilator alarms may indicate high airway pressures.

Treatment involves emergent decompression by insertion of a large-bore catheter or needle into the second or third intercostal space in the midclavicular line (**Appendix 11**). This should not be delayed for radiographic confirmation. Decompression is both diagnostic and therapeutic and will restore blood pressure and vital signs to normal. The insertion of the catheter or needle must be followed by placement of a thoracostomy tube to prevent recurrence of the pneumothorax.

B. Conversion from Negative to Positive Intrathoracic Pressure

Normal intrathoracic pressure is slightly negative in relation to the atmosphere. When positive pressure ventilation is initiated, intrathoracic pressure becomes positive. As intrathoracic pressure rises, right atrial pressure rises, and the intravascular pressure gradient for return of blood from the large extrathoracic veins into the right heart decreases. As a result, systemic venous return to the heart is reduced. Left ventricular preload, stroke volume, cardiac output, and blood pressure may then decrease in sequence. The deleterious effects on cardiac output and blood pressure are exacerbated

by underlying intravascular volume depletion.

Treatment of this common complication involves volume resuscitation using rapidly infused fluid boluses, in aliquots of 10 or 20 mL/kg, to raise extrathoracic venous pressure and increase venous return to the right heart until the blood pressure increases. Oxygen saturation should be monitored to avoid overly aggressive fluid resuscitation. Use of ventilation techniques that incorporate high mean airway pressure may exacerbate the deleterious hemodynamic consequences of positive pressure ventilation.

C. Auto-PEEP

Auto-PEEP occurs when the lungs do not empty completely before the initiation of a subsequent breath. Caused by either obstruction to outflow of gas or a need for an excessively long inspiratory time, the combination of ventilator settings and patient physiology results in an inadequate expiratory time (**Figure 5-4**). Excessive auto-PEEP may increase intrathoracic pressure and cause hypotension because of decreased venous return to the heart. Although auto-PEEP may occur in any patient, those with obstructive airway disease are particularly predisposed to this condition. Immediate relief of auto-PEEP can be achieved by briefly disconnecting the ventilator circuit from the ETT. Assessment and treatment of auto-PEEP is then performed.

D. Acute Myocardial Ischemia/Infarction

Factors that can increase myocardial oxygen demand include: stress from acute respiratory failure, intubation, and the transition to positive pressure ventilation; β-adrenergic medications (ie, epinephrine [adrenaline], dobutamine); and some anesthetic agents (ketamine). In older children and adults with inadequate cardiac reserve, this may lead to acute myocardial ischemia and subsequent hypotension.

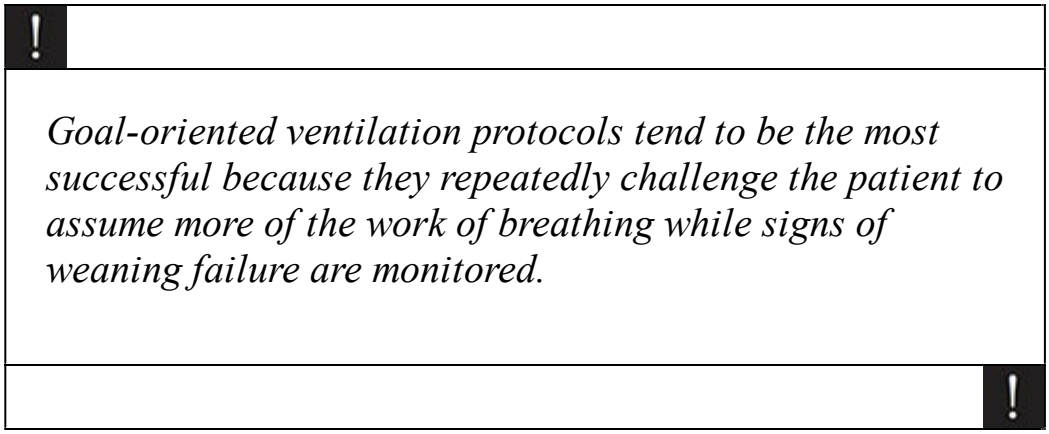
XI. WEANING FROM MECHANICAL VENTILATION

Although beyond the scope of this fundamental course, no discussion of mechanical ventilation would be complete without a short review of current approaches to weaning the patient from the ventilator. This process should be considered as soon as pulmonary function begins to improve. Decisions concerning weaning are separate from decisions concerning extubation.

During weaning, the patient gradually takes on more of the function of gas exchange while the ventilator contributes less to the patient's minute ventilation. The circumstances that required mechanical ventilation support must be resolving. In addition, the patient must actively participate in the process, and objective and subjective criteria must be set to monitor the success or failure of the process. Factors that influence weaning from mechanical ventilation include the patient's level of alertness, need for sedative medication, respiratory drive, and the time elapsed since the patient's most recent episode of respiratory or hemodynamic decompensation.

Several approaches to weaning have been proposed for adults and children. They include the demonstration of the patient's readiness for further weaning and ultimately for extubation, and almost universally include a spontaneous breathing trial. The conditions under which the trial takes place may vary, but an important component is the use of pressure support to prevent the patient's work of breathing from being hampered by the resistance of the ventilator circuit. In small children with an ETT <3.5 mm, unsupported spontaneous breathing may be akin to breathing through a straw. If respiratory muscles are deconditioned, the child's effort may be insufficient to overcome tube resistance. Fatigue and respiratory distress may ensue unless pressure support is added.

The rate at which a patient is weaned from mechanical ventilation is based on a number of factors, including the patient's physical condition and the duration of mechanical ventilation. A patient with normal lungs who is placed on mechanical ventilation for general anesthesia during an elective surgical procedure can be weaned and extubated rapidly, which anesthesiologists often do in the operating room. This is in contradistinction to the patient who is intubated for several days or weeks for severe systemic illness, who requires a more gradual, closely monitored weaning regimen. Conditions should be optimized before weaning is initiated; this includes adjusting sedatives and discontinuing neuromuscular blocking medications, optimizing nutrition and ensuring a positive nitrogen balance, and optimizing fluid and acid-base status. Patients with severe metabolic alkalosis (often the result of aggressive diuretic therapy) should have their acid-base status normalized to ensure optimal respiratory drive.



Goal-oriented ventilation protocols tend to be the most successful because they repeatedly challenge the patient to assume more of the work of breathing while signs of weaning failure are monitored.

Once weaning has begun, a change in vital signs – such as an increase in heart rate or respiratory rate, and an increase or decrease in blood pressure – may indicate a patient's inability to tolerate the process. Clinical signs of fatigue, including paradoxical breathing or use of accessory muscles of respiration, diaphoresis, or agitation, suggest that the patient is not ready to be weaned further. Patients who become air hungry are easily recognized by their inability to attend to anything other than their next breath. The patient who becomes more tachypneic, develops retractions, and requires more oxygen to maintain the same saturations is failing to wean and should be placed back on a higher level of support. Arterial blood gas values are an objective indicator of how well a patient is tolerating the weaning. A need for more than 50% F_{IO_2} to achieve a Pao_2 >60 mm Hg (>8.0 kPa) or a $Paco_2$ >50 mm Hg (>6.7 kPa) usually indicates that the patient is not a candidate for further weaning. A chest radiograph should be obtained during the final stages of weaning to ensure there are no significant contraindications to extubation, such as atelectasis or pneumothorax.

Key Points

Mechanical Ventilation

- The primary goals of invasive and noninvasive mechanical ventilation are to support ventilation and oxygenation and to reduce the work of breathing while ensuring patient comfort.
- Noninvasive modes of ventilation may be adequate intervention for patients with mild to moderate respiratory disease and no significant comorbid conditions.
- Patients receiving noninvasive ventilation should be intubated if their condition worsens or if there is no significant response to NIV modes within 1 to 2 hours.
- Various types of breaths and modes of invasive mechanical ventilation are available to facilitate synchrony between the patient and the ventilator.
- The following guidelines may be used when initiating volume-control ventilation: F_{IO_2} of 1.0; an age-appropriate rate, usually between 12 and 24 breaths/min; V_T of 6 to 8 mL/kg that results in adequate chest rise (V_T of 4-6 mL/kg for lungs with low compliance); and PEEP between 2 and 5 cm H_2O. The inspiratory time, usually 0.5 to 1 second, is based on the patient's age and disease process.
- The complex interactions between inspiratory pressures, I:E ratio, F_{IO_2}, and PEEP must be appreciated to predict the potential beneficial and harmful effects in each patient.
- The primary determinants of oxygenation are F_{IO_2} and mean airway pressure, whereas minute alveolar ventilation (respiratory rate and V_T) primarily affects CO_2 exchange. In the patient with acute lung injury or ARDS, PEEP becomes an additional independent determinant of oxygenation.
- The importance of closely monitoring the patient during mechanical ventilation cannot be overstressed. This includes appropriate setting of and attention to ventilator alarm functions, continuous pulse oximetry, frequent bedside assessment of the patient, and obtaining arterial blood gases and chest radiographs as needed.
- Weaning from mechanical ventilation is an active process that should be started as soon as the indications for ventilation have begun to resolve. Close monitoring during the weaning process is required to avoid any additional stress and to ensure patient comfort.

Suggested Readings

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DIAGNOSIS AND MANAGEMENT OF SHOCK

Objectives

- Define shock.
- Identify the 5 major categories of shock and the typical presentations.
- Determine the initial steps in the evaluation and management of a child in shock.
- Discuss the principles of fluid resuscitation for pediatric shock.
- Outline the physiologic effects of vasopressors and inotropic agents.
- Recognize the importance of a best-practices, evidence-based approach to care through the design and implementation of institution-specific recognition, resuscitation and stabilization, and performance bundles.

I. INTRODUCTION

Shock, a common problem in acute care settings, may be simply described as inadequate delivery of oxygen and other essential substrates to vital organs. Circulatory and cellular alterations manifest as clinical phenotypes and result in a variety of metabolic abnormalities stemming from impaired oxygen transport.

Oxygen delivery (Do_2), which is compromised in shock states, is directly related to the arterial oxygen content (Cao_2) and cardiac output (CO). Cardiac output is a function of the heart rate and stroke volume, the amount of blood ejected from the left ventricle with each beat. Cao_2 represents the amount of oxygen bound to hemoglobin plus the very small amount of oxygen dissolved in arterial blood.

$$Do_2 = Cao_2 \times CO$$

$$CO = \text{heart rate} \times \text{stroke volume}$$

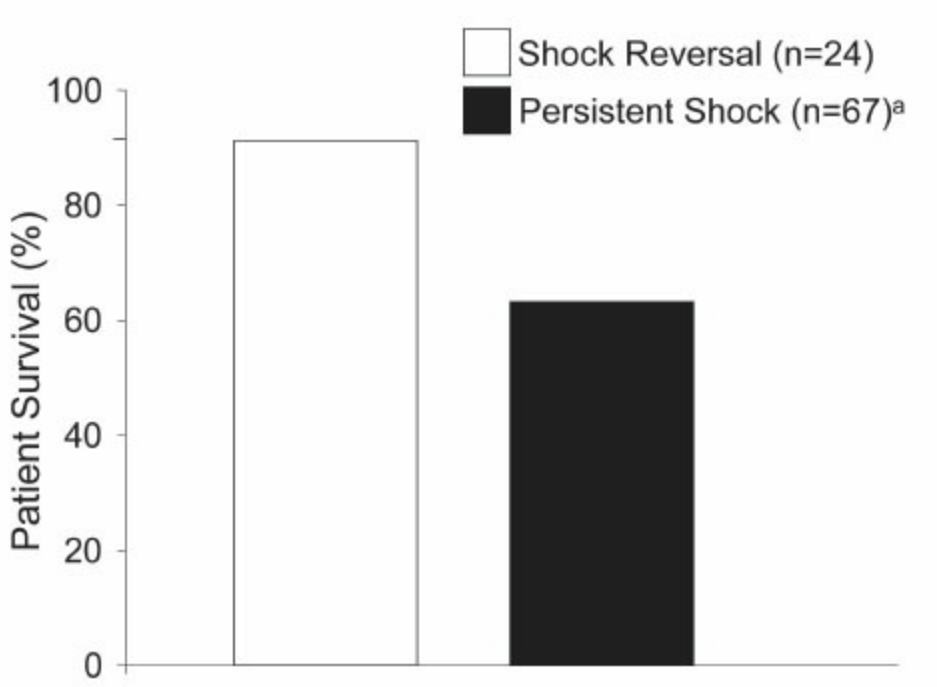
$$Cao_2 = (\text{hemoglobin} \times 1.34 \times Sao_2) + (0.003 \times Pao_2)$$

Stroke volume is a function of *preload*, defined as the volume of blood in the ventricular cavities at the end of diastole, the *afterload* or resistance to ventricular ejection, and *myocardial contractility*, the force exerted by the myocardium.

Inadequate cardiac output can be corrected by ensuring optimal preload, afterload, and contractility and, in some cases, by correcting an abnormally high or low heart rate. In addition, oxygen delivery can be increased by optimizing hemoglobin oxygen saturation and by raising the circulating hemoglobin concentration with packed red blood cell transfusion.

Shock-related mortality has decreased dramatically over the last few years largely due to implementation of evidence-based guidelines, which emphasize prompt diagnosis and rapid, serial goal-directed interventions designed to reverse the signs of shock and restore vital signs to normal, age-appropriate values. Han et al demonstrated that the reversal of shock within the first hour of a patient's presentation to a community hospital was associated with dramatic improvements in mortality rate and functional outcome (**Figure 6-1**). Subsequent studies have demonstrated reductions in morbidities such as acute kidney injury, in mortality rate, and in resource utilization with adherence to evidence-based guidelines and protocolized resuscitation bundles.

Figure 6-1. Survival Rates of Pediatric Patients with Shock Reversal Versus Persistent Shock

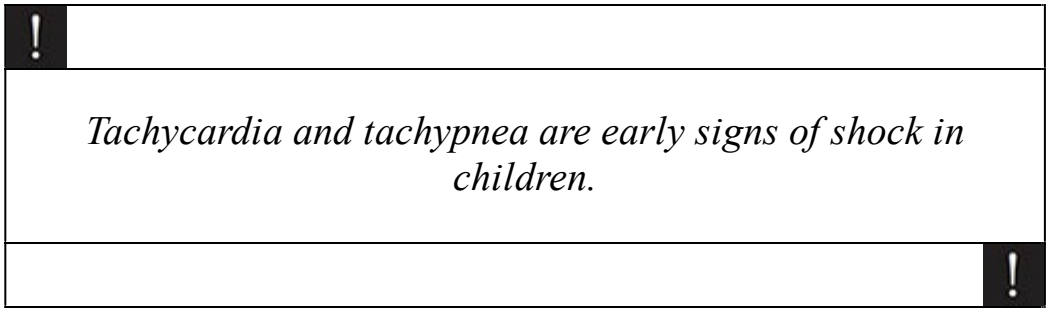


a P <0.05.
Shock reversal from resuscitative efforts by community hospital physicians resulted in 96% survival versus 63% survival among patients who remained in a persistent state of shock.
Data taken from Han YY, Carcillo JA, Dragotta MA, et al. Early reversal of pediatric-neonatal septic shock by community physicians is associated with improved outcome. *Pediatrics*. 2003;112:793-799.

Developmental differences between pediatric and adult patients strongly influence management decisions: although the cardiac rate and contractility usually increase in acutely ill adult patients, stroke volume is relatively fixed in infants, so that any decrease in heart rate critically diminishes cardiac output. Infants and children also have higher baseline heart rates, which limit the ability to improve cardiac output by increasing the heart rate.

Systemic vascular resistance can increase significantly in the acutely ill child. Therefore, blood pressure may remain in a falsely reassuring range until the sudden onset of decompensated shock. Elevated systemic vascular resistance also compromises cardiac output, particularly in the presence of hypovolemia or decreased myocardial function.

Early shock in pediatric patients often has subtle presentations. Tachycardia and tachypnea may be the only abnormalities. Failure to detect (within the first 5 minutes of presentation) and reverse these phenomena (within the first hour) may allow a compensated, easily manageable shock state to progress to irreversible organ dysfunction.



II. TYPES OF SHOCK

The 5 main categories of shock are hypovolemic, distributive, cardiogenic, obstructive, and anemic.

A. Hypovolemic Shock

A previously healthy 2-month-old child is brought to the hospital because of severe dehydration. He has been having voluminous stools for the last 2 days. He is lethargic, with a heart rate of 220 beats/min and a blood pressure of 60/50 m Hg. The anterior fontanelle and eyes are sunken. He has thready pulses and cold extremities, with markedly delayed capillary refill. Venous access proves difficult; an intraosseous needle is placed in his tibia, and he receives 60 mL/kg normal saline within an hour of presentation. His heart rate decreases to 140 beats/min, his blood pressure improves to 76/40 mm Hg, and he starts making good eye contact. Capillary refill and distal pulses improve.

Detection

– What is this child's diagnosis?

Intervention

– What is the first step in resuscitating this child?

Reassessment

The boy is able to sit up and interact appropriately with caregivers after receiving isotonic crystalloid, 60 mL/kg, in the first hour.

– How will you determine if he needs more fluid resuscitation and/or other therapeutic interventions?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

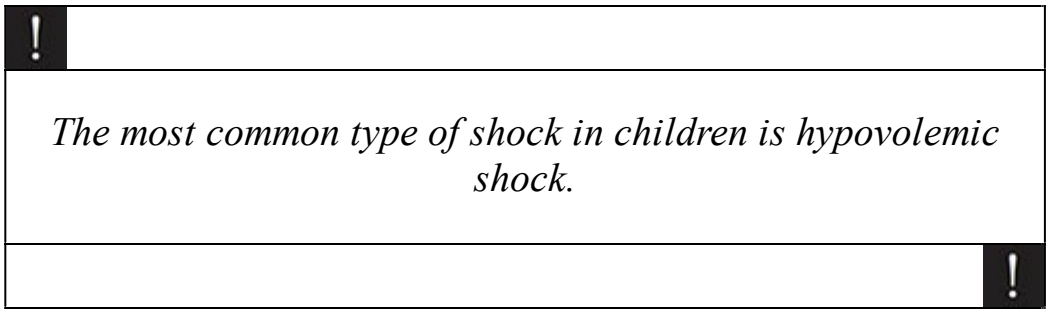
– Where is the best place to manage the care of this patient?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

Hypovolemic shock, the most common type of shock seen in children, occurs when circulating intravascular volume is compromised to the point where adequate cardiac output and tissue perfusion can no longer be maintained. The initial response to hypovolemia is the activation of peripheral and central baroreceptors, in turn promoting catecholamine-mediated vasoconstriction and tachycardia. These mechanisms can maintain adequate circulation and blood pressure even after the acute loss of as much as 30% of the circulating blood volume. Acute losses in excess of 30% result in a clinically obvious state that may rapidly cause life-threatening organ compromise.



The most common cause of hypovolemia is diarrhea, the leading cause of child death worldwide. Hypovolemia may develop rapidly in patients unable to mitigate ongoing losses due to concurrent vomiting or inadequate parenteral correction.

Children with traumatic injuries and significant or ongoing blood losses may rapidly progress to hemorrhagic shock. These patients typically present with tachycardia, a narrowed pulse pressure, delayed capillary refill, orthostatic changes, and in late stages, frank hypotension. They commonly show signs of decreased end-organ perfusion, such as altered mentation and decreased urine output, which may be delayed when high osmotic loads are present. Oliguria may not be evident until severe shock has developed.

B. Cardiogenic Shock

A 16-year-old previously healthy girl complains of feeling unwell and collapses in her bedroom. Initial evaluation in the emergency department reveals the following: heart rate, 160 beats/min; respiratory rate, 36 breaths/min; blood pressure, 60/50 mm Hg; cool, ashen, clammy extremities and clear lungs. The liver is not palpable. She seems to have an enlarged cardiac silhouette on chest radiograph. She appears confused and has no urine output. The girl is placed on oxygen and is given a normal saline bolus of 10 mL/kg.

Detection

– What physical exam findings suggest the correct diagnosis?

– What additional studies should be done?

Intervention

– What are the immediate interventions needed?

– Do any initial interventions now need to be changed?

Reassessment

The heart rate decreases to 140 beats/min; her blood pressure remains unchanged, but her color is improved. She is given another saline bolus of 5 mL/kg and dopamine is initiated (10 μg/kg/min). Her blood pressure improves to 110/70 mm Hg, and she starts to produce urine. Her general appearance improves, and she is transferred to the pediatric ICU.

– Is the current treatment strategy effective?

– What else does this patient need? What other therapeutic interventions may be necessary?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How should the treatment strategy be implemented?

– Who is to do what and when?

Cardiogenic shock is caused by myocardial pump dysfunction with decreased cardiac output. Patients with cardiogenic shock are usually – but not always – tachycardic, with a narrow pulse pressure and cool extremities. Myocardial dysfunction leading to cardiogenic shock may be caused by a systemic inflammatory response syndrome elicited by sepsis or injury, infections (including viral), and autoimmune and familial cardiomyopathies.

Sequelae of hypoxic-ischemic events, dysrhythmias with impaired coronary perfusion, and acidosis may be present on arrival at the emergency department or may develop during hospitalization. Abnormalities in heart rate, such as severe tachycardia in patients with supraventricular tachycardia, may cause florid heart failure in a few hours. Profound bradycardia in patients with complete heart block may also precipitate end-organ dysfunction.

Cardiogenic shock must be considered in the differential diagnosis when acute illness is encountered in young infants. Infants and young children with myocardial dysfunction due to supraventricular tachycardia, primary cardiomyopathy, or structural congenital heart disease may display subtle signs

of heart failure, such as irritability, fatigue, vomiting, and poor feeding (diaphoresis, tiring easily or needing an extended period to complete a feeding).

Infants with congenital cardiac lesions that are dependent on the patency of the ductus arteriosus (ductus-dependent lesions) may also present with cardiogenic shock in the first 2 months of life. A significant gradient in the blood pressure measured in the upper extremities compared to the lower extremities is typical of left-sided, ductus-dependent congenital cardiac lesions, which may rapidly cause life-threatening shock and organ dysfunction. An infusion of prostaglandin E_1 should be started immediately and continued until echocardiography definitively excludes a ductus-dependent lesion.

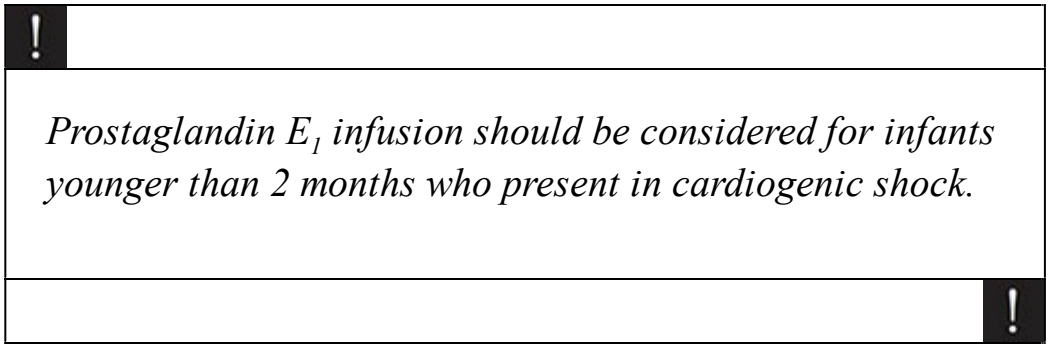
Acute decompensated heart failure typically presents with 1 of 4 phenotypes, determined by their clinical features: wet, dry, warm, or cold. Patients with a "wet" phenotype typically are hypervolemic and exhibit signs of pulmonary and/or systemic congestion. Respiratory distress, wheezing, rales, and a gallop may be heard; hepatomegaly and peripheral edema may be present. Jugular venous distention may be difficult to visualize in the young and in obese patients. When measured, high atrial and ventricular filling pressures are present.

Patients with a "dry" phenotype may have normal or low intravascular volume (preload), manifested by poor skin and end-organ perfusion. These patients sometimes have relative hypovolemia and thus may respond to small, carefully titrated fluid boluses.

In the "warm" phenotype, patients have normal or near-normal end-organ perfusion. Patients with the "cold" phenotype have cool, clammy extremities indicative of a low cardiac-output state. They typically have more profound and difficult-to-correct impairments in end-organ function.

Regardless of the phenotype, treatment of cardiogenic shock centers on optimizing intravascular volume through cautious use of fluids if there is a question of relative hypovolemia and with diuretics if the child appears hypervolemic. Close attention to fluid management is critical, as overzealous fluid resuscitation in a child in "wet and warm" heart failure may lead to a "wet and cold" shock. Myocardial contractility can be augmented with inotropic agents; afterload reduction can be achieved with the appropriate use of vasodilators.

General supportive measures are of paramount importance. Myocardial oxygen consumption may be minimized by achieving optimal filling pressures and the appropriate use of vasoactive medications. The control of fever, pain, and anxiety also contributes to reducing stress on the heart and may be very beneficial.



C. Distributive Shock

A 16-year-old boy is brought to the hospital after sustaining a blunt injury to his upper back during football practice. He is alert, seems flushed, and has a heart rate of 60 beats/min and blood pressure of 70/30 mm Hg. His hands and feet are warm. No urine output is noted despite placement of a Foley catheter. His heart rate drops to 48 beats/min; he complains of feeling nauseous and becomes lethargic.

Detection

– What are the important clues to this patient's underlying physiology?

– What are common causes of distributive shock?

Intervention

– What are the immediate interventions needed?

– How should this patient be stabilized?

Reassessment

Aggressive fluid resuscitation is begun, with normal saline (60 mL/kg) given within the first hour. A phenylephrine infusion is started, and the boy's blood pressure and general appearance rapidly improve.

– Is the current treatment strategy effective?

– What ongoing therapeutic interventions will be needed?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Distributive shock is caused by inappropriately decreased systemic vascular resistance with redistribution of blood flow away from vital organs. This type of shock may be observed in patients with early septic shock, anaphylaxis, toxic ingestions, and spinal cord injuries, or those who have received spinal or epidural anesthesia. Children with distributive shock typically have a flushed appearance, warm extremities with bounding pulses, and tachycardia with a wide pulse pressure. Capillary refill may be instantaneous.

Distributive shock due to spinal cord injury may occur without significant compensatory rises in heart rate (relative bradycardia). Treatment should focus on reversal of the underlying etiology if possible,

rapid expansion of intravascular volume, and infusion of vasopressor medications with predominantly α-adrenergic activity, such as phenylephrine or norepinephrine (noradrenaline), to increase systemic vascular tone. Children with toxic ingestions (eg, tricyclic agents) are prone to ventricular dysrhythmias. Sodium bicarbonate is used to overcome the drug-induced sodium channel blockade underpinning these dangerous dysrhythmias.

D. Obstructive Shock

A 12-year-old previously healthy boy is an unrestrained front-seat passenger involved in a motor vehicle accident. He is alert, complaining of diffuse chest discomfort and dyspnea. His heart rate is 150 beats/min and blood pressure is 80/60 mm Hg. He is taking frequent, shallow breaths. He has tracheal deviation towards the left. Breath sounds are asymmetric; heart tones seem distant. The abdomen is soft. Pulses are thready, and the extremities are cold. His condition abruptly worsens, and his blood pressure becomes undetectable.

Detection

– What are the important clues to this child's underlying physiology?

– What are common causes of obstructive shock?

– What are the clinical findings that suggest pleural and pericardial collections?

Intervention

– What are the immediate interventions needed?

– How should this child be stabilized?

Reassessment

A chest tube is placed as fluid resuscitation with isotonic crystalloid progresses via the push-pull method. His vital signs improve just as cardiopulmonary resuscitation is about to begin. A chest radiograph shows his chest tube to be in position, and his trauma evaluation is continued.

– What general management principles must be observed?

– Is the current treatment strategy effective?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Obstructive shock is relatively uncommon in pediatrics, so a high index of suspicion is necessary. It results from extrinsic forces on the intrathoracic great vessels or cardiac chambers. Cardiac output is limited despite normal intravascular volume and myocardial contractility. Common causes of obstructive shock include pleural collections, such as pneumothoraces or hemopneumothoraces. Pulmonary embolism, a rare cause of obstructive shock, should be considered in children at risk, including those with hypercoagulable states or long bone fractures. Tamponade physiology may occur when pericardial effusions form rapidly and limit ventricular filling.

Clinical clues to the diagnosis of obstructive shock include tachycardia, cool extremities with delayed capillary refill, a narrow pulse pressure, distended neck veins, distant heart tones, and asymmetric breath sounds. Tracheal or mediastinal deviation may be seen with large pleural collections. Treatment centers on the prompt reversal of the underlying cause.

Volume resuscitation with isotonic crystalloids or colloids is an essential temporizing measure. Drugs that decrease systemic vascular resistance, such as benzodiazepines, opioids, or propofol, should be used with extreme caution until the cause of the obstructive shock is reversed because severe hypotension or cardiac arrest may occur. Dramatic improvement usually follows evacuation of a tension pneumothorax or drainage of a pericardial effusion.

E. Anemic Shock

A 22-month-old previously healthy girl is brought to the emergency department following several days of fever, rhinorrhea, decreased activity, and rash. Examination reveals an ill-appearing, small, pale, alert, but very quiet child. Her heart rate is 180 beats/min, respiratory rate is 28 breaths/min, and blood pressure is 101/46 mm Hg. She has conjunctival pallor, a “slapped cheek” appearance, and a systolic ejection murmur at the left upper sternal border. The liver is palpated ~ 2 cm below the right costal margin. There are no other focal findings on exam. Her history reveals that she is a picky eater and drinks at least 32 ounces of whole milk each day.

Detection

– What are the important clues to this child’s underlying physiology?

– What disease process or combination of diseases might present in this way?

Intervention

– What immediate interventions are needed?

– What interventions should be avoided?

Reassessment

Laboratory testing reveals the following results: white blood cell count, 1500 mm^3; hemoglobin 1.9 g/dL; hematocrit, 7.6%; and platelets, 300,000 mm^3. Results of blood chemistry, lactate

dehydrogenase, uric acid, and urine analysis are all normal. A fecal occult blood test is positive. Additional lab studies are suggestive of iron deficiency anemia. The result of a parvovirus DNA polymerase chain reaction test is positive.

– How should this child's anemia be addressed?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will this information be disseminated?

– What education needs to be provided to the family before the patient is discharged from the hospital?

Teamwork

– How will the treatment strategy be implemented?

Children with chronic severe anemia have high cardiac output failure. Research done in the malaria belt of Africa shows that these children may be harmed by fluid boluses but benefit from IV fluids administered at a maintenance infusion rate and administration of blood transfusion for hemoglobin levels <5 g/dL. Acute anemic shock responds better to blood transfusion than to crystalloid fluid boluses.

F. Septic Shock

A previously healthy adolescent complains of severe malaise and shortness of breath. He describes the onset of an influenza-like illness and left knee swelling 2 days before presentation. He is alert, febrile, tachycardic (160 beats/min), and ill-appearing, with mild respiratory distress. He has brisk capillary refill, bounding pulses, and a blood pressure of 86/30 mm Hg.

Detection

– What are the important clues to this patient's underlying physiology?

– What is the cause for this septic shock?

Intervention

– What are the immediate interventions needed?

– What is the correct volume and period of time over which to administer the fluid bolus?

– What are the management priorities for pediatric shock?

Reassessment

The patient receives 500 mL of normal saline over 1 hour and is then transferred to the pediatric ICU (PICU). On arrival, he is noted to be obtunded and dyspneic, with extremely poor perfusion and

undetectable blood pressure. The crash cart is brought to the bedside; the patient is placed on high-flow oxygen, and isotonic crystalloids are rapidly administered in 20 mL/kg aliquots using a push-pull setup. An epinephrine infusion is started via existing access as preparations are made for intubation and placement of a central venous line.

– What general shock management principles must be observed?

– What are common pitfalls in the management of pediatric shock?

– Why is "cold shock" difficult to reverse?

Effective Communication

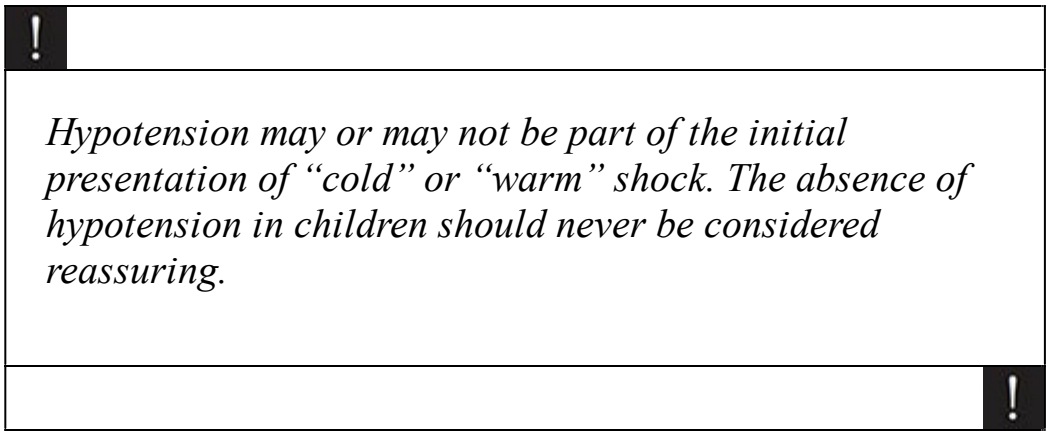
– When the patient's clinical status changes, who needs to know and how will this information be disseminated?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

The inflammatory triad of fever, tachycardia, and vasodilation is commonly seen in children with benign acute febrile illnesses; septic shock, on the other hand, should be suspected in children with these features in the presence of altered mentation, especially decreased interaction with parents or healthcare providers. Signs of septic shock, even in the presence of normal systolic blood pressure, include: hypothermia, hyperthermia, abnormalities in perfusion manifested as mottling, cool extremities, delayed capillary refill, diminished or a "warm" phenotype with bounding pulses, wide pulse pressure, and decreased urine output.



Hypotension may or may not be part of the initial presentation of "cold" or "warm" shock. The absence of hypotension in children should never be considered reassuring.

Patients with septic shock often manifest elements typical of hypovolemic, distributive, and cardiogenic shock. They can have relative or absolute hypovolemia, abnormal (and variable) vascular tone, maldistribution of blood flow, and myocardial dysfunction. Acidosis, coagulopathy, and metabolic and endocrine derangements culminate in abnormalities in energy utilization at the subcellular level. Capillary leak syndromes develop quickly and may further complicate patient management (**Table 6-1**).

Table 6-1	POSSIBLE ETIOLOGIES OF SHOCK

Hypovolemic Shock
- Fluid losses: gastrointestinal disorders (acute gastroenteritis, pancreatitis), renal dysfunction
- Blood loss: hemorrhage resulting from trauma or coagulopathy
- Capillary leak: burns, intestinal ischemia caused by volvulus, intussusception, or necrotizing enterocolitis

Septic Shock
- Bacterial
- Viral
- Fungal
- Rickettsial
- Parasitic

Distributive Shock
- Neurogenic shock: head injury, spinal cord injury
- Anaphylaxis
- Drug toxicity
- Adrenal insufficiency: congenital adrenal hyperplasia

Obstructive Shock
- Aortic stenosis, coarctation of the aorta, or other left-heart obstructive lesions (infants <6 mo)
- Unrecognized pneumothorax
- Cardiac tamponade
- Pulmonary embolism

Cardiogenic Shock
- Congenital heart lesions
- Myocardial dysfunction (systolic or diastolic): ischemic heart disease (Kawasaki disease), cardiomyopathies
- Dysrhythmia: supraventricular tachycardia
- Metabolic disorders
- Drug poisoning

Some children with septic shock may initially display a hyperdynamic, high cardiac output state characterized by vasodilatation, tachycardia, bounding pulses, brisk capillary refill, and a flushed appearance, a pattern commonly called *warm shock*.

Children with dopamine-resistant shock have clinical features of low cardiac output, including tachycardia; altered mental status; poor peripheral pulses; mottled skin; and prolonged capillary refill. This presentation has often been referred to as *cold shock*.

Cardiac output can change from high to low rapidly as septic shock progresses. These patients go from a flushed, warm, hyperdynamic state to one marked by hypoperfusion with tachypnea, tachycardia, hypotension, prolonged capillary refill, weak pulses, and metabolic (lactic) acidosis.

Treatment goals include prompt identification of shock, rapid reversal of the cardiovascular dysfunction with adequate intravascular volume expansion, early intubation, vasoactive and inotropic support, and control of the infectious organism with antimicrobials and source control interventions. Immunosuppression should be discontinued in patients suspected to have serious systemic infection.

III. EVALUATION OF SHOCK

A systematic, well-coordinated, context-appropriate approach to children presenting with features of shock should be developed by each institution. This is facilitated by the use of recognition, resuscitation, stabilization and performance bundles.

The evaluation of any illness or injury in all patients must include thorough initial and serial

assessments of the airway, breathing, and circulation (ABCs), with complete sets of vital signs, including blood pressure measurements, which are commonly missed in infants. Shock should be suspected within the first 5 minutes of evaluation of any ill-appearing child presenting to the emergency department or deteriorating in the inpatient setting. Fluctuations in levels of consciousness are important clinical clues to be followed using the conventional or modified Glasgow Coma Scale.

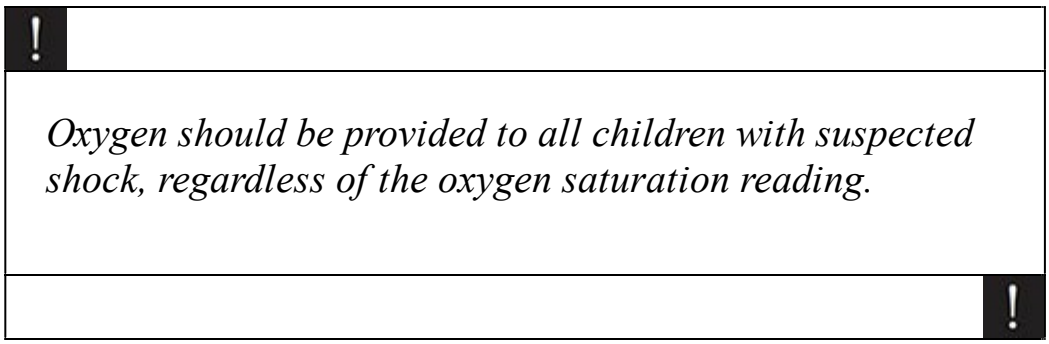
A. Assessment of Airway, Breathing, and Circulation

Use of the DIRECT algorithm (**Chapter 1**) facilitates serial assessments of a child's vital signs, mentation, and perfusion. Strong teamwork with closed-loop communication is essential in the systematic, comprehensive approach to pediatric shock. Subtle abnormalities may quickly progress to hemodynamic collapse.

Some institutions utilize a "shock clock" to ensure that basic interventions are completed in a timely fashion. Some institutions call "time zero" upon discovery of tachycardia and tachypnea in ill-appearing children. Recognition of shock, initiation of high flow oxygen, and vascular access should be started within the first 5 minutes of evaluation.

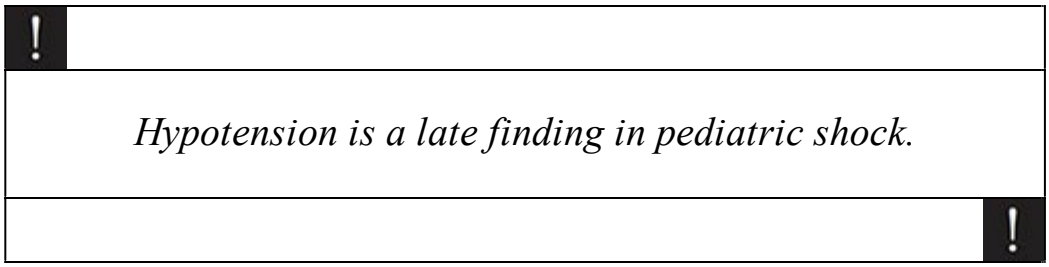
Airway patency, respiratory rate, respiratory pattern, and work of breathing should be noted. All children with suspected shock should receive supplemental oxygen, regardless of their baseline oxygen saturations. Positioning should be optimized to ensure airway patency and minimize work of breathing. Continuous pulse oximetry is usually well tolerated, even by irritable children, and is mandatory in the evaluation and monitoring of any sick patient. Hypoxemia or the presence of a poor pulse oximeter signal is an ominous sign warranting immediate attention.

High-flow nasal oxygen may be helpful in patients with mildly to moderately increased work of breathing; a non-rebreathing face mask can help preoxygenate children who may subsequently require airway intervention. These patients require close, serial observations. Children displaying severe respiratory distress on presentation, and those failing to improve or deteriorating despite these measures, require assisted ventilation preoxygenation (and careful bag-mask ventilation if needed) until preparations have been made for endotracheal intubation. Any patient with a rapidly declining Glasgow Coma Scale score (**Chapter 15**) or a score of ≤8 or less is at risk for respiratory arrest, severe hypoxemia, and severe hypercarbia. The airway should be secured as soon as possible by the most experienced practitioner available. Judicious use of induction agents helps avoid hemodynamic collapse.



Severe bradycardia (heart rate <90 beats/min in an infant) and severe tachycardia (heart rate >180

beats/min in ill-appearing infants <1 year or >140 beats/min in older children and adolescents) are markers of the severe systemic compromise that may precede circulatory collapse. Severe tachycardia in an obviously ill child may be quickly followed by hypotension and cardiac arrest. Any abnormality in heart rate should be confirmed with a rhythm strip or an electrocardiogram to define the underlying rhythm. A pediatric intensivist or cardiologist should be contacted for optimal management of dysrhythmias in children.



Hypotension is a late finding in pediatric shock, given children's capacity to increase systemic vascular resistance. They may be normotensive or even hypertensive in the presence of established shock. Blood pressure may fall precipitously in these patients. Failure to diagnose shock in a timely manner—before the onset of hypotension—may allow progression to an uncompensated and possibly irreversible state (**Table 6-2**).

Table 6-2 MINIMUM ACCEPTABLE BLOOD PRESSURE VALUES FOR ILL-APPEARING CHILDREN

Age Group	**Systolic/Diastolic Values (mm Hg)**
Term neonate	60/30
Infant	80/40
Child	90/60
Adolescent	100/70

Classified using US Department of Health and Human Services. *The Fourth Report on Diagnosis, Evaluation and Treatment of Hypertension in Children and Adolescents*. Bethesda, MD: National Institutes of Health; 2005. NIH Publication 05-5267.

Fever is common but not universal in pediatric patients with serious infections. In fact, hypothermia may be noted in infants and in septic patients of any age, making core temperature monitoring extremely important. Infants commonly react to a cold environment with apnea and bradycardia. Surface temperatures and capillary refill, easily reproducible indicators of perfusion, should be assessed in a central location and in an extremity that has not been exposed to a cold environment. Significant difference in the temperatures of the torso and the toes suggests impaired perfusion.

Capillary refill is complete within 2 to 3 seconds under normal conditions. Abnormalities in refill time give important clues about the etiology of the circulatory derangement and can help assess response to interventions. These measures, along with the quality of central and peripheral pulses, are markers that must be followed closely as resuscitation progresses.

B. Blood Glucose

A bedside (point-of-care) assessment of blood glucose is essential upon presentation and at

appropriate intervals in all acutely ill children. Infants are particularly vulnerable to hypoglycemia given their high metabolic rates and limited glycogen reserves. Failure to promptly diagnose and treat hypoglycemia may result in permanent neurologic disability (**Chapter 8**). Repeat assessments of glucose levels should be performed during resuscitation and stabilization efforts.

C. History and Physical Examination

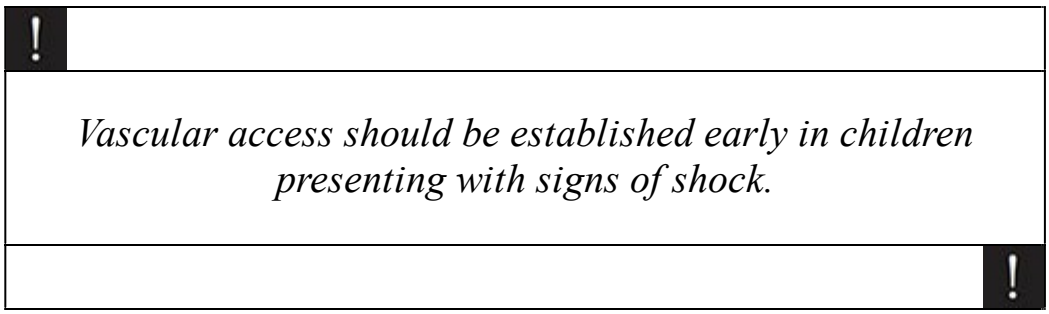
A patient's history and physical examination will reveal important clues to the etiology of shock. The lung fields should be auscultated to detect new rales or asymmetry of breath sounds. A gallop rhythm may indicate underlying heart disease or hypervolemia due to excessive fluid administration. Palpation of the liver edge below the costal margin – particularly when it is blunt rather than sharply defined – can indicate hypervolemia or cardiac failure. Rapidly evolving purpuric or petechial rashes may suggest an infectious origin. A careful secondary survey can reveal previously missed injuries in children with unclear or unknown histories.

IV. MANAGEMENT OF SHOCK

All ill-appearing patients presenting with abnormal vital signs suggestive of shock should be placed on high-flow oxygen via face mask (running at least 6 L/min to ensure adequate sweeping of carbon dioxide from the mask) within the first 5 minutes, while their initial evaluation is performed. Peripheral or intraosseous vascular access should be secured within the first few minutes of evaluation, preferably within this initial 5-minute timeframe. Initial fluid resuscitation with up to 60 mL/kg isotonic crystalloid, control of the airway, provision of appropriate vasoactive infusions, and broad-spectrum antibiotic therapy are priorities to be addressed within the first 60 minutes of presentation.

A. Vascular Access

Vascular access must be obtained as soon as possible. IV cannulas may be difficult to place in acutely ill children with poor peripheral perfusion. Common sites for catheter placement include the dorsum of the hands, the saphenous vein just anterior to the medial malleolus, and the veins in the antecubital fossae. Initial volume resuscitation may be readily accomplished using scalp veins in infants. Care should be taken to avoid cannulation of the temporal artery when these veins are utilized. Umbilical venous access is an option for neonates if a skilled provider is available to perform the procedure.



The external jugular vein may be accessible to cannulation; however, care should be used in the presence of respiratory distress or increased intracranial pressure to avoid further physiologic compromise with positioning. External jugular cannulation sites must be monitored for infiltration and removed as soon as more secure access is obtained, given the potential for infiltration with movement and the risks of embolism and thrombosis.

Professionals caring for sick children must have a low threshold for placement of intraosseous needles for initial or supplemental vascular access. Intraosseous access is a simple, safe, and effective measure to be considered early in the care of children with inadequate vascular access. Intraosseous needles can be successfully placed in children of all ages; although the proximal and distal tibia are the most common sites utilized in children, other sites, such as the distal femur and proximal humerus, should be considered when indicated (**Appendix 2**).

Central venous catheters are helpful in complex resuscitations and should be placed by healthcare providers with expertise in this procedure when transfer to a tertiary center may be delayed. Proper control of the airway, correct positioning, adequate analgesia, local anesthesia, and strict sterile technique with full barrier precautions are required (**Appendix 12**).

Subclavian venous access is more treacherous in children than in adults, and a pneumothorax or vascular injury may be rapidly fatal in patients with preexisting hypovolemia. Therefore, subclavian cannulation should generally be avoided in children with shock, particularly in those with suspected coagulopathy.

The right internal jugular vein can be cannulated using the posterior approach, which entails entering the vein as it courses beneath the sternocleidomastoid muscle. This is most easily performed by piercing the skin at the lateral border of the muscle immediately above the external jugular vein. The needle is advanced under the muscle towards an imaginary line that connects the point of entry with the sternal notch and the left nipple, with the head turned to the left and the neck slightly extended with a roll under the shoulders. Other approaches to the internal jugular vein, such as the central or anterior, are facilitated by ultrasonography. Although the left internal jugular vein may also be accessed with these techniques, this approach carries the added risk of injury to the thoracic duct.

Cannulation of the femoral vein is relatively easy and safe. Hemodynamically significant retroperitoneal hematoma or injury to the femoral artery may occur rarely. Infants are at higher risk for vascular injury, particularly if the provider is inexperienced. The vein is readily accessed medial to the femoral pulse. Ultrasound guidance is recommended for central vascular access in all patients when available.

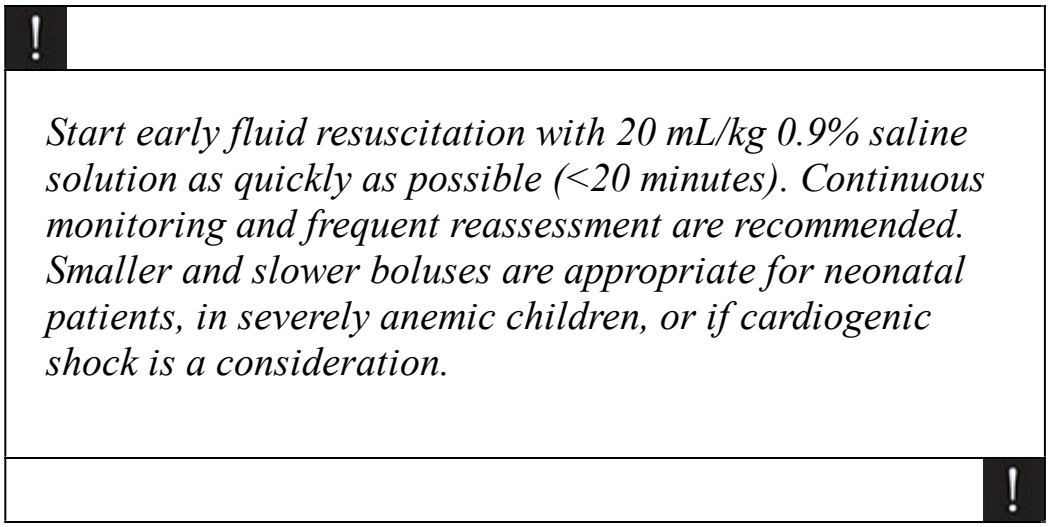
Central venous pressure monitoring is advised, either on a continuous basis or as a trend every few hours if the lumen is required for infusion. Low central venous pressure is indicative of absolute hypovolemia in the ill, supine patient. High venous pressures suggest the opposite, with the caveat that they may be increased by high intrathoracic pressures, pneumothorax, or pericardial effusion, underscoring the importance of clinical correlation. Some patients with underlying cardiac disease require higher filling pressures; echocardiography and subspecialty consultation are helpful in such cases.

Arterial cannulation with continuous monitoring is recommended for critically ill children with

persistent shock requiring ongoing interventions. Peripheral access is preferred, as injury to the femoral artery may occur. Important information provided by this intervention includes the continuous display of heart rate, blood pressure, and pulse pressure, trends that inform patient management. The presence of a sloped upstroke suggests myocardial dysfunction with poor contractility (**Appendix 15**).

B. Fluid Resuscitation

Since hypovolemia is the most common cause of pediatric shock, it follows that most children with shock require volume resuscitation. This underscores the importance of vital sign monitoring and the clinical exam. Trends should be towards normalization of vital signs; the presence or onset of rales, hepatomegaly, or worsening respiratory distress or perfusion suggest hypervolemia and myocardial dysfunction.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Start early fluid resuscitation with 20 mL/kg 0.9% saline solution as quickly as possible (<20 minutes). Continuous monitoring and frequent reassessment are recommended. Smaller and slower boluses are appropriate for neonatal patients, in severely anemic children, or if cardiogenic shock is a consideration.
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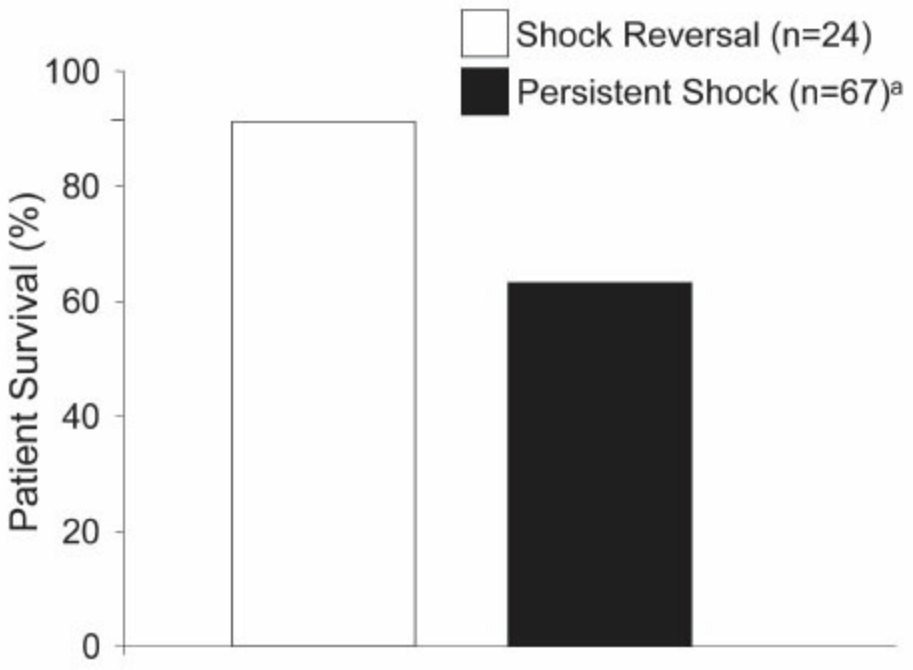
Large amounts of fluids are commonly required for resuscitation in hypovolemia or sepsis, due to children's higher ratios of surface area to volume and the potential for ongoing losses. Children are less likely to have underlying comorbidities than adults and are generally better able to handle the large amounts of fluids administered during the course of resuscitation. Very large amounts of fluid may be required, particularly in those with septic shock or significant ongoing losses. Improved outcomes have been observed when at least 60 mL/kg of rapid intravascular fluid expansion is given within the first hour of treatment to children with septic shock (**Figure 6-2**).

Figure 6-2. Effects of Early Fluid Resuscitation in Pediatric Patients With Septic Shock



[a]Increased survival compared to all other groups.
Number of survivors and nonsurvivors among pediatric patients with septic shock stratified in groups according to the volume of fluid resuscitation received within the first hour of presentation to the emergency department.
Data taken from Carcillo JA, Davis AL, Zaritsky A. Role of early fluid resuscitation in pediatric septic shock. *JAMA*. 1991;266:1242-1245.

Only isotonic solutions should be used in the rapid intravascular volume expansion of children in shock. Normal saline (0.9% NaCl) is readily available and should be administered in aliquots of 20 mL/kg as quickly as possible (preferably <20 minutes) using peripheral IV or intraosseous access. Smaller fluid boluses should be used for neonates or if cardiogenic shock is suspected. Resistance to flow is significant when small central venous catheters are utilized, underscoring the value of peripheral and intraosseous access.

Although vigorous fluid resuscitation does not correlate with adverse pulmonary or neurologic outcomes in children, there are some concerns when large amounts of normal saline are administered for resuscitation. One is that normal saline, like other resuscitation fluids, begins to redistribute to the extravascular space in as little as 1 hour, contributing to dilution of plasma proteins, edema, and anasarca with serosal effusions, including ascites and pleural collections. Hyperchloremic metabolic acidosis is common but transient. Lactated Ringer solution (Hartmann solution) is used extensively in some countries and can be effective in the management of moderate to severe shock, particularly if albumin solutions are unavailable. It is less likely to produce hyperchloremic acidosis than normal saline but must be used with caution in patients at high risk for hyperkalemia.

To mitigate such problems some experts advocate the use of 5% albumin, which remains longer in the intravascular space. Another approach is to use isotonic crystalloid initially and to continue volume resuscitation using 5% albumin if more than 40 to 60 mL/kg are required to normalize perfusion. One meta-analysis suggested that volume expansion with crystalloids may be advantageous for pediatric patients with gastroenteritis and injuries, including burns and traumatic brain injury. Volume expansion with colloids may be preferable in severe sepsis. Packed red blood cell transfusion is the resuscitative fluid of choice in severe anemia.

Smaller aliquots of resuscitation fluid (5-10 mL/kg) should be administered to neonates and to children with known or suspected myocardial dysfunction or heart disease. Frequent reassessment with serial vital signs and repeated physical exam (specifically assessing for rales, hepatomegaly, and worsening edema) is mandatory in this situation to avoid delayed resuscitation and precipitation of congestive heart failure.

A common but dangerous error is to infuse fluid boluses over 1 hour. The infusion pumps routinely used for medication and IV fluids are not effective for rapid resuscitation, even when set at "999" mL/h. The simplest measure is to interpose a 3-way stopcock (tap) into the IV line, so that fluids may be pulled from the bottle or bag (taking care not to aspirate and inject an air bolus, which could be fatal) and then administered via the peripheral or central venous line or intraosseous needle. Serial reassessment is important to help guide the initial fluid resuscitation, which should be continued until adequate perfusion is achieved, the child's vital signs have returned to target values, and general appearance has improved. Use of the DIRECT algorithm is recommended.

Hemodilution associated with crystalloid or colloid boluses may compromise oxygen delivery in the presence of severe anemia, underscoring the need for packed red blood cell transfusion in these circumstances. Anemic children with severe malaria, respiratory distress, or neurologic dysfunction who are receiving care for shock in resource-limited settings, such as sub-Saharan Africa, appear to benefit from early transfusion of packed red blood cells as opposed to multiple boluses of isotonic crystalloid or colloid.

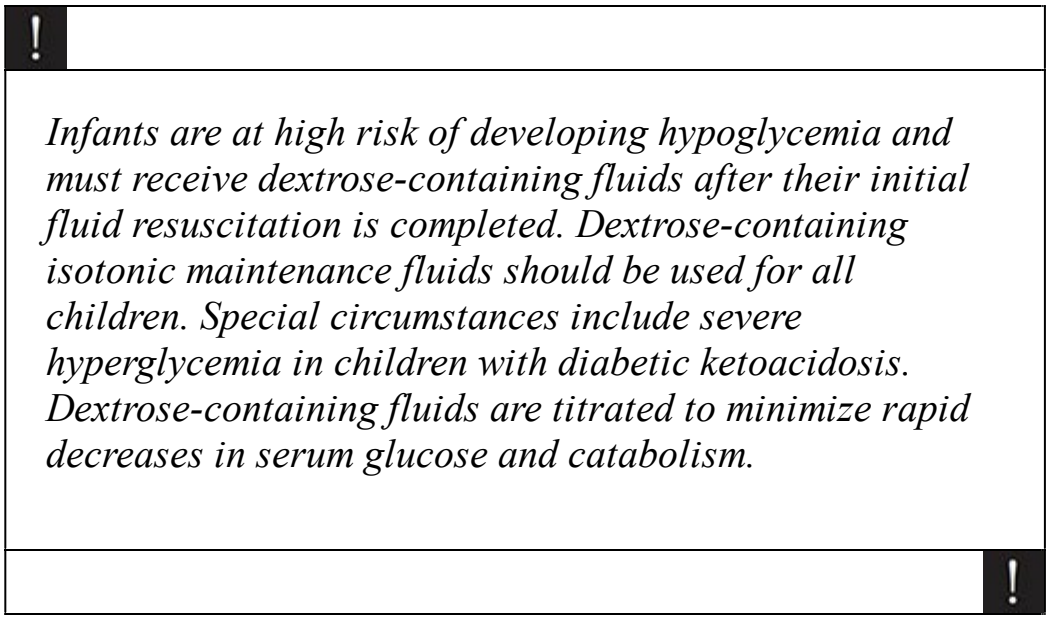
Fluid resuscitation should be continued until clinical improvement or clinical evidence of a hypervolemic state (suggested by new rales, a gallop rhythm, or hepatomegaly) is seen. Continued bolus administration of fluids at this point will cause excessive myocardial wall forces and depress myocardial performance. Diuretic therapy with small doses of furosemide (0.3-0.5 mg/kg, with a maximum of 20 mg in a furosemide-naïve patient) may be considered if hypervolemia is suspected in an otherwise stabilizing child. This strategy may result in improved myocardial performance and enhanced urine output, and may mitigate oliguric renal failure if acute tubular necrosis has developed. Higher doses of diuretics may be required in certain children, but it is prudent to administer a small dose first, particularly in the presence of hemodynamic instability.

Children who required resuscitation and are felt to be close to a normovolemic state (neither "wet" nor "dry") may be placed on maintenance fluids, adding boluses of normal saline, 5% albumin, or appropriate blood products as required. Infants are at high risk of developing hypoglycemia and therefore should receive dextrose-containing fluids, 5% to10% dextrose with appropriate sodium and potassium supplementation, and frequent (hourly, if needed) bedside glucose monitoring.

A useful formula for calculating the infusion rate of maintenance fluids is to provide 4 mL/kg/h for the first 10 kg of body weight, plus 2 mL/kg/h for each additional kilogram up to 20 kg, with 1 mL/kg/h for each additional kilogram of body weight thereafter. Adult-sized children can be managed with maintenance infusions of 125 to 150 mL/h. An alternative is to calculate the body surface area and provide 1500 mL/m² as a 24-hour infusion, to be supplemented with isotonic or colloid boluses as required. Hyponatremia is common in critically ill patients. In patients of all ages, 5% dextrose and normal saline-based fluids can be used for the initial maintenance fluid.

Hypoglycemia diagnosed at presentation should be managed with boluses of a 10% dextrose solution

(5 mL/kg). For older children, 25% glucose can be used as required to achieve a serum glucose level >100 mg/dL. This is preferred to the infusion of large volumes of dextrose-containing crystalloids, which may be relatively hypotonic and carry the risk of inducing osmotic diuresis.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Infants are at high risk of developing hypoglycemia and must receive dextrose-containing fluids after their initial fluid resuscitation is completed. Dextrose-containing isotonic maintenance fluids should be used for all children. Special circumstances include severe hyperglycemia in children with diabetic ketoacidosis. Dextrose-containing fluids are titrated to minimize rapid decreases in serum glucose and catabolism.
!

Patients at risk for neurologic injury due to trauma or a hypoxic-ischemic insult are also best managed with isotonic IV fluids. Although normal saline is commonly used, hypoglycemia is possible and may cause severe permanent injury. Hyperglycemia is commonly observed in acute stress and usually resolves during resuscitation without specific intervention. Aggressive treatment of hyperglycemia is treacherous during patient transfers. Regular insulin infusions must be used with great caution when absolutely indicated, started as low continuous infusions, and monitored closely at the bedside, at hourly intervals, for appropriate management of hypo- or hyperglycemia.

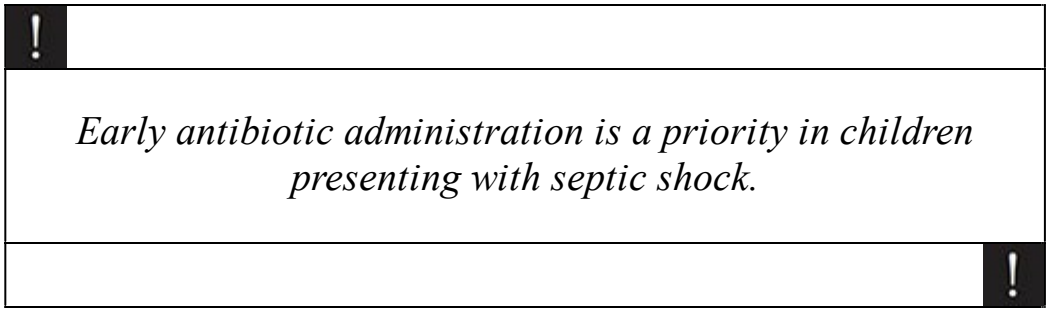
Blood products may be required in special circumstances, such as traumatic injury or surgical losses in critically ill patients. The risk of hypotension precludes the rapid infusion of fresh frozen plasma. Platelets and cryoprecipitate may be indicated in the presence of thrombocytopenia and hypofibrinogenemia, emphasizing the importance of obtaining appropriate laboratory studies as soon as possible during the resuscitation.

C. Antibiotics

Ampicillin and gentamicin are widely available and appropriate first choices for neonatal patients. Nafcillin, a third-generation cephalosporin, and vancomycin broaden coverage to other common pathogens, such as *Staphylococcus aureus*, *Streptococcus pneumoniae*, group A streptococci, and *Neisseria meningitidis*. Metronidazole, cefoxitin, or piperacillin/tazobactam is necessary in patients at risk for anaerobic infections, such as those with complex wounds or abdominal sepsis.

Although blood and urine cultures are important in determining the cause of infections, antibiotics should be given within the first hour of presentation and should never be delayed by difficulty in securing appropriate samples. Morbidity and mortality rates increase with each passing hour of delayed antimicrobial therapy.

In patients with bacteremia and sepsis, a massive release of cytokines from lysis of bacteria follows even the first dose of an antibiotic and can result in hemodynamic instability in 60 to 90 minutes. This can occur during interfacility transport to a tertiary care center and has been noted especially in meningococcemia and other Gram-negative infections.



D. Vasoactive and Inotropic Support

Vasoactive and inotropic infusions are helpful in fluid-refractory shock, defined as persistence of abnormal vital signs despite appropriate fluid resuscitation, typically 60 mL/kg given in the first hour of diagnosis. Vasoactive infusions augment perfusion pressures, particularly after hypovolemia has been corrected. These infusions should be started via existing peripheral venous or intraosseous access and can be maintained in this fashion until central venous access has been secured. Although vasoactive infusions in theory may worsen end-organ perfusion pressures in the presence of hypovolemia, they should not be delayed while high-volume fluid resuscitation is in progress.

Adults display typical and relatively static hemodynamic patterns, but these patterns are less predictable and can change rapidly in children. Invasive hemodynamic monitoring with an arterial catheter and a central venous catheter should be used to provide continuous measurements and establish trends in cardiovascular performance. Serial assessments of venous saturation in the superior vena cava are useful in guiding management and have been shown to reduce mortality rates in children with septic shock.

1. Epinephrine

Epinephrine (adrenaline) has many important physiologic effects from the activation of α- and β-receptors. Clinically relevant chronotropy, increased cardiac contractility, and increased systemic vascular resistance are observed, particularly at higher doses, making it a useful agent in the management of children with shock. Epinephrine is the drug of choice for cold septic shock, anaphylactic shock, and cardiogenic shock in patients with hypotension.

A typical starting dose is 0.05 to 0.1 μg/kg/min, titrated to clinical effect, while being mindful of its potential to decrease systemic vascular resistance at the low range and to increase it at higher doses. This drug should be administered as a continuous infusion into a reliable peripheral vein until a central venous catheter is in place.

Epinephrine may increase myocardial oxygen consumption and cause a decrease in splanchnic perfusion. It may decrease coronary perfusion pressures, particularly in the presence of hypovolemia or severe tachycardia. These are important considerations in older children, who may be at greater

risk for myocardial ischemia and dysrhythmias (**Table 6-3**).

Table 6-3	HEMODYNAMIC EFFECTS OF COMMONLY USED VASOACTIVE AND INOTROPIC AGENTS				
Agent	**α_1**	**β_1**	**β_2**	**D_1**	**V_1**
Dopamine[a]	Vasoconstriction; ↑ SVR, PVR	Inotropy; chronotropy	Vasodilatation	Vasodilatation (renal)	
Dobutamine		Inotropy			
Epinephrine[b] (adrenaline)	Vasoconstriction; ↑ SVR, PVR	Inotropy; chronotropy			
Norepinephrine (noradrenaline)	Vasoconstriction; ↑ SVR, PVR	Inotropy (minor)			
Vasopressin	Potentiates	Potentiates			Vasoconstriction
Non-receptor-mediated					
Milrinone	Inotropy, lusitropy, vasodilatation				

D_1, dopamine receptor; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance.

[a]Dose-related effects: at low doses, D_1-receptor effects might predominate; at intermediate doses, β_1- and β_2-receptor effects predominate; and at high doses, α_1-receptor effects predominate.

bDose-related effects: at low doses, β_1- and β_2-receptor effects predominate; at high doses, α_1-receptor effects predominate on peripheral vasculature.

Adapted with permission. © 2006 Elsevier. Smith L, Hernan L. Shock states. In: Fuhrman BP, Zimmerman J, eds. *Pediatric Critical Care*. 3rd ed. Philadelphia, PA: Mosby; 2006:394-410.

2. Norepinephrine

Norepinephrine (noradrenaline) differs from epinephrine (adrenaline) in its strong α-receptor selectivity, resulting in increased vascular tone with minimal effect on the heart rate. This, in turn, translates into higher coronary and splanchnic perfusion pressures, and possibly decreased end-organ dysfunction. The typical starting dose is 0.05 to 0.1 μg/kg/min, and the agent is quickly titrated to the desired effect. Norepinephrine is the drug of choice in warm septic shock and is a good choice for spinal cord shock if phenylephrine is unavailable.

3. Dopamine

Dopamine had for decades been considered to be the first-line vasoactive medication for treatment of shock. Some clinicians are now moving toward epinephrine and norepinephrine as first-line agents. Dopamine has dose-related clinical effects. Renal vasodilation is thought to occur at low infusion rates, although this not clinically relevant. At intermediate doses of 5 to 10 μg/kg/min, β_1 and β_2 effects predominate, resulting in inotropy, chronotropy, and vasodilatation, which may be advantageous in some situations. Higher infusion rates, typically in excess of 10 μg/kg/min, result in α_1-receptor activation, with increases in systemic and pulmonary vascular resistance.

Relatively small doses of dopamine, such as 5 μg/kg/min, may be all that is needed to provide inotropic support for normotensive children. However, the drug should always be started at higher

doses, typically 10 µg/kg/min in children who are hypotensive, to avoid possible β-receptor–mediated vasodilation. Increasing the dose of dopamine beyond 20 µg/kg/min is not beneficial, because a major portion of its effect is the release of epinephrine/norepinephrine from stores. The absence of a response to escalating doses of dopamine indicates that the patient is likely to be in a catechol-depleted state. Infants may be relatively insensitive to dopamine, requiring a directly acting catecholamine, such as epinephrine or norepinephrine. Clinically significant increases in pulmonary vascular resistance have been described in some neonates when dopamine and other vasopressor medications are used (**Table 6-3**). Vasoactive agents commonly used in stabilization of critically-ill children (**Table 6-4**).

Table 6-4	SUGGESTED INFUSION DOSES FOR COMMONLY USED VASOACTIVE AND INOTROPIC AGENTS	
Agent	**Inotropic**	**Pressor**
Dopamine	2-15 µg/kg/min	>12 µg/kg/min
Dobutamine	2.5-20 µg/kg/min	
Epinephrine (adrenaline)	0.05-0.5 µg/kg/min	0.1-1 µg/kg/min
Norepinephrine (noradrenaline)		0.05-1 µg/kg/min
Vasopressin		0.5 mU/kg/min
Non-receptor-mediated		
Milrinone	0.25-0.75 µg/kg/min	

Adapted with permission Jain S, Vaidyanathan B. Digoxin in management of heart failure in children: Should it be continued or relegated to the history books?. *Ann Pediatr Card* 2009;2:149-52.

4. Vasopressin

Vasopressin is a potent vasoconstrictor that is useful in the management of shock with decreased systemic vascular resistance. It exerts its hemodynamic effects via the V_{1a}-receptor, promoting an increase in intracellular calcium, and thus restoring systemic vascular tone. Vasopressin has been associated with improved blood pressure and urine output in patients with catecholamine-refractory vasodilatory shock and may allow weaning of catecholamines. Although a pediatric dose has not been established, some experts recommend starting with 0.5 mU/kg/min (0.0005 U/kg/min) for children.

Septic shock may contain features of hypovolemic, distributive, cardiogenic, anemic, and obstructive shock, underscoring the importance of serial examinations to guide interventions. The standard adult dose of 0.03 or 0.04 U/min should not be exceeded.

5. Dobutamine

Dobutamine has significant inotropic effects given its preferential activation of β-receptors. Although it may have a role in the management of infants with mild hypoperfusion, typically in doses of 5 µg/kg/min, higher doses may be associated with unacceptable tachycardia and vasodilation. This agent is not as helpful in older children, particularly in adolescents with septic shock, in whom a decrease in systemic vascular resistance and an increase in heart rate may compromise coronary

perfusion.

Dobutamine is a useful agent when ventricular dysfunction is suspected or documented. This drug—commonly used following cardiac transplantation, surgery to repair congenital heart defects, and myocardial revascularization, and when myocardial function is significantly depressed—has a limited role in the contemporary management of pediatric septic shock.

6. Milrinone

Milrinone is commonly utilized in pediatric critical care for its inotropic and lusitropic effects in patients with myocardial dysfunction in the presence of a high systemic vascular resistance and low cardiac output. It has a long elimination half-life and duration of action and requires dose adjustment in patients with renal and hepatic failure. Significant and prolonged hypotension may be observed, particularly when a loading dose(s) in excess of 0.3 μg/kg/min are employed or in the presence of renal or hepatic dysfunction. Milrinone is best initiated after admission to the PICU, with patients receiving continuous intravascular monitoring.

7. Nitroprusside and Nitroglycerin

Nitroprusside and nitroglycerin have vasodilator effects and much shorter half-lives than milrinone, making them more appropriate for short-term use if a vasodilator is considered essential before transfer to a tertiary center. These agents can be utilized at very low doses, such as 0.5 μg/kg/min, to achieve desired decreases in systemic vascular resistance (reduce afterload) to "unload" the left ventricle in cardiogenic shock as long as blood pressure is adequate. Adequate volume loading with isotonic crystalloids or colloids, and the immediate availability of a vasopressor (such as epinephrine or norepinephrine) are required for use.

E. Endotracheal Intubation and Mechanical Ventilation

Mechanical ventilation, with careful preparation for intubation and meticulous attention to tidal volumes and pressures, may reduce the work of breathing, optimize oxygen transport, and improve hemodynamic performance by reducing left ventricular afterload. Subsequent planned and emergent procedures are significantly facilitated by a secure airway.

Due to the potential for deterioration during intubation in patients with shock, the most experienced person available should perform this procedure, preferably after volume resuscitation is well under way. A vasopressor infusion should already be initiated or be readily available at the bedside for immediate use.

Preoxygenation via a non-rebreathing face mask or an anesthesia circuit is extremely important. A rapid-sequence intubation with appropriate precautions should be utilized in patients with recent oral intake or those at risk for delayed gastric emptying. Care must be taken not to distort anatomical landmarks if cricoid pressure is applied. Patients in shock may exhibit delayed gastric emptying due to compromised splanchnic circulation. Gastrostomies may be present in chronically ill children and should be opened to air or gravity drainage. A nasogastric tube may be placed if excessive gastric distention has already occurred, but it is best placed after the airway has been secured to prevent

vomiting before intubation.

Given infants' fixed myocardial contractility and their dependence on heart rate for adequate cardiac output, drug premedication should include atropine to prevent vagally mediated bradycardia. Atropine improves intubating conditions in patients of all ages by minimizing airway secretions and possibly decreasing airway tone. There are no contraindications to using atropine in appropriate doses, even in the presence of tachycardia, so it should be considered for all children requiring emergent intubation.

Drugs that may decrease systemic vascular resistance, such as midazolam or morphine, must be avoided in children with shock. Although many sources cite midazolam doses of 0.1 mg/kg, that amount may have significant deleterious hemodynamic effects in tenuous patients. Midazolam should be titrated carefully in small aliquots of ≤0.05 mg/kg, with the intention of using it as an adjuvant rather than the primary agent for intubation. Small doses of fentanyl (1-2 μg/kg) are preferred to morphine, which causes substantially more hemodynamic effects.

Ketamine is the drug of choice for intubating patients in shock because of its dissociative anesthetic properties and its minimal adverse effects on hemodynamics. An IV dose of 1 to 2 mg/kg is usually effective and facilitates bag-mask ventilation before neuromuscular blocking agents are administered. Although ketamine has been traditionally avoided in patients with known or suspected intracranial hypertension, this is no longer considered to be a contraindication since several studies have shown that the cerebral perfusion pressure is maintained without significant rise in intracranial pressure. This agent is still avoided in some patients with known hydrocephalus.

Ketamine is generally safe and effective in emergencies. It may be given intramuscularly (3 mg/kg) with atropine in patients who do not have vascular access. Atropine is commonly given with ketamine to mitigate sialorrhea and is preferred to glycopyrrolate in emergent procedures given its vagolytic effect. The use of small doses of benzodiazepines to decrease the incidence of emergence phenomena following ketamine administration has been called into question. While ketamine is considered to have potential neuroprotective effects in traumatic brain injury, there is concern regarding neuroapoptosis in young infants and children.

Etomidate may cause adrenal insufficiency and has been associated with increased risk of death in adults in septic shock. It should be used with caution because of the potential for adrenal insufficiency. Both ketamine and etomidate have a shorter duration of action than commonly used neuromuscular blocking agents. Consequently, they need to be redosed or a longer acting sedative and/or analgesic should also be administered once the airway is secured and the hemodynamics are adequate.

Although propofol is used widely in adult medicine, it commonly causes hypotension in children, particularly in the presence of hypovolemia or myocardial dysfunction. Therefore, it is not recommended in children with shock, particularly if transport is required.

Neuromuscular blockade may be necessary to ensure patient safety and ventilator synchrony, but it should be utilized only with adequate sedation and analgesia by individuals with excellent airway management skills. Rocuronium and vecuronium are neuromuscular blocking agents frequently utilized in securing the airway. Atracurium and cisatracurium may have a relatively higher propensity

to cause histamine release with consequent hypotension and bronchospasm. They are best used in the PICU for specific indications, such as in patients with severe hepatic or renal dysfunction. Succinylcholine is still preferred by some clinicians due to its rapid (<60 seconds) onset and short duration (5-10 minutes) of action. It should be avoided in children at risk for hyperkalemia, such as those with renal failure, extensive burns, or crush injury, those who are known to have a myopathy, or patients who have been bed-bound for extensive periods.

All patients should be preoxygenated and initially ventilated with an fraction of inspired oxygen of 1.0; this can be titrated as the child stabilizes to maintain saturations of 95% or above during transport to the PICU. Oxygen should be used with caution in patients with ductus-dependent lesions because oxygen can stimulate ductal closure.

The familiar mechanical ventilation modes should be selected. The synchronized intermittent mandatory ventilation mode, with either volume control or pressure control, is commonly used. Most patients with shock and normal pulmonary mechanics can be ventilated with standard tidal volumes of 6 to 8 mL/kg. Ventilation with pressure-controlled modes should aim for adequate chest rise; the amount of pressure needed to produce an adequate rise and ventilate small infants with normal lung compliance may be very small. Close attention to chest rise (in all modes) and attainment of peak inspiratory pressures (optimally <30 cm H_2O in volume control modes) may help decrease secondary lung injury (including pneumothorax) and the hemodynamic repercussions of mechanical ventilation.

Adequate positive-end expiratory pressure helps optimize ventilation and perfusion. Use of at least 4 to 5 cm H_2O is recommended for all patients; higher levels may be needed in children who have severe respiratory failure. An inspiratory time of 0.6±0.2 seconds can be used for most patients, selecting longer inspiratory times for larger children. An inspiratory/expiratory (I:E) ratio of 1:2 is suitable for most patients with shock.

Respiratory rates of 24 to 30 breaths/min may be sufficient for small infants; adult-sized children are usually ventilated with rates of 12 breaths/min, and smaller children receive rates up to 20 breaths/min. Capnography and venous or arterial blood gas levels, when available, should guide ventilator management.

For more information, please see the chapter on mechanical ventilation (**Chapter 5**). Advanced mechanical ventilation strategies for specific problems, such as the presence of congenital heart disease or persistent pulmonary hypertension in the newborn, should be attempted only after consultation with the tertiary center.

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A tachypneic, dyspneic, and agitated child with increased work of breathing may have 30% to 50% of the cardiac output supplying the inspiratory muscles and diaphragm. Safely sedating, intubating, and mechanically ventilating such a patient can conserve cardiac output to supply vital organs, such as the brain and myocardium.

F. Endocrine/Hormonal Support

As noted previously, infants and young infants with shock of any etiology are at high risk of developing hypoglycemia. Hyperglycemia is a common response to the stress of acute illness; it may subside as shock resolves. Insulin therapy is necessary in children with diabetes mellitus, preferably using a low dose (0.05 U/kg/h) with hourly glucose checks and an additional glucose-containing fluid source at the bedside, particularly if interfacility transport is required.

Adrenal insufficiency must be suspected in children with fluid-refractory shock. Risk factors include congenital adrenal insufficiency, known pituitary or adrenal abnormalities, recent steroid exposure (within the past year), purpura fulminans, head injury, abdominal injury, exposure to etomidate as an induction agent, or exposure to megestrol or ketoconazole in chronically ill children. Hydrocortisone (usually in doses equivalent to 100 mg/m²/d) may facilitate stabilization. Provision of at least 20 mg IV hydrocortisone may obviate the need for mineralocorticoid replacement with fludrocortisone.

Hypothyroidism requiring replacement may occur in acutely ill children with trisomy 21, in infants after surgery for congenital heart disease, and in the presence of severe brain injury. It also may be seen following administration of corticosteroids in patients with severe shock.

Hypocalcemia, which is common in the young but occur in patients of all ages, may be addressed with calcium gluconate or chloride infusions.

G. Control of Acidosis

Metabolic acidosis, frequently offset by respiratory alkalosis in the initial stages of shock, may become evident as a child's condition worsens. Optimal management includes addressing underlying causes, such as hypovolemia and abnormal organ perfusion, and using volume resuscitation and vasoactive infusions as required. Adequate ventilation and oxygenation should be ensured by the use of cautious dosing of sedation and analgesia agents, adding neuromuscular blockade only if absolutely necessary.

Sodium bicarbonate is indicated primarily for patients with hyperkalemia or intoxication with sodium channel-blocking drugs, but it may also be considered in the management of selected profoundly acidemic (pH <7), adequately ventilated patients. Because the use of sodium bicarbonate may increase intracellular acidosis, this treatment is no longer routinely recommended. Buffering agents such as tromethamine (given slowly, generally 3-5 mL/kg over an hour) may be helpful in extreme situations, with the caveat that they can worsen hypokalemia, hyperkalemia, and hypoglycemia. These agents should be avoided in the presence of established renal dysfunction.

H. Laboratory Evaluation

A point-of-care bedside glucose assay should be performed at presentation in any child who is

acutely ill, because significant time may elapse between sampling and the report of the laboratory results. The initial evaluation should also include a complete blood count, complete chemistry profile, ionized calcium, lactate, blood gases, and coagulation testing as warranted by the child's condition and a presumptive cause of shock. Serial measurements of superior vena cava saturations may be helpful in following the child's response to therapy and in complementing serial physical examinations. B-type natriuretic peptide and troponin levels may be indicated in children with features of myocardial dysfunction.

Blood typing and cross-matching should be performed as soon as possible to expedite the availability of blood products that may be required for ongoing resuscitation.

Lumbar puncture should be deferred until a child has stabilized. Positioning and the stress of lumbar puncture may result in significant deterioration and cardiopulmonary arrest, particularly in the presence of increased intracranial pressure, coagulopathy, or limited reserve, conditions that may be found in patients who have shock. This is particularly important when the airway has not been secured. Lumbar puncture findings will not affect the initial management of septic shock.

Additional laboratory studies, such as measurement of ammonia, serum amino acids, and urine organic acids, may be indicated in patients suspected of having inborn metabolic errors. These problems are usually detected in early infancy but may present in older infants and children. Toxicology screens are important for any child presenting with shock and unexplained encephalopathy. Assays for endocrine dysfunction, including cortisol levels and thyroid function tests, should be considered in specific circumstances, although they should not delay the administration of hydrocortisone (1 mg/kg, up to 50 mg IV every 6 hours) and fludrocortisone in children with suspected adrenal insufficiency.

I. Radiographic Studies

A chest radiograph is essential to the complete evaluation of any child presenting with shock. Heart size provides important clues. Most children with hypovolemia have a small or normal-appearing heart. A large cardiac silhouette suggests myocardial dysfunction, hypervolemia, or pericardial effusion. The position of all tubes and lines should be noted. A more extensive survey that includes the lateral cervical spine and pelvis is required in patients who may have sustained traumatic injuries. Bedside ultrasound scanning can provide important clinical clues. Careful consideration should be given to securing the airway before moving the child for specialized imaging (eg, computed tomography, magnetic resonance imaging) because deterioration may occur rapidly in settings unprepared for cardiac arrest.

V. GENERAL CARE

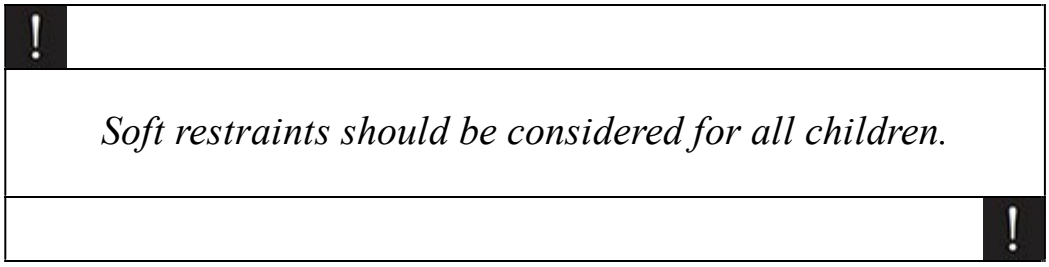
Bundled protocolized care has reduced mortality rates in a variety of settings. Important components of initial resuscitative care include complete initial and serial assessment of the airway, breathing, and circulation. Supplemental oxygen must be provided immediately. Adequate vascular access must be secured within the first 5 minutes of evaluation. It may be necessary to control the airway to ensure

adequate oxygenation and ventilation, to normalize perfusion, and to restore vital signs to the threshold values for age. Antibiotic therapy should start within the first hour if there is any suspicion of sepsis.

Appropriate monitoring includes continuous pulse oximetry, electrocardiography, frequent (every 5 min) blood pressure measurements, hourly temperature readings, hourly urine output measurement via an indwelling urinary catheter, and frequent assessments of glucose and ionized calcium. Arterial and central venous catheters allow for continuous monitoring of hemodynamic variables and intermittent sampling of superior vena cava saturations to trend the patient's progress.

A point-of-care glucose measurement should be obtained hourly in patients at high risk of hypoglycemia, especially infants. Indicators of end-organ dysfunction, coagulation, and gas exchange should be evaluated at least every 6 hours.

A systematic, head-to-toe approach, guided by the DIRECT algorithm, should be employed during serial examinations while awaiting transfer. Appropriate analgesia, control of fever with acetaminophen, sedation, and neuromuscular blockade decrease oxygen consumption. Sedation and analgesia can be safely and effectively accomplished using low-dose, titrated infusions or small, titrated intermittent doses of benzodiazepines and fentanyl or ketamine (**Chapter 20**).



Continued meticulous attention to maintaining optimal preload, afterload, and contractility is essential. An adequate hemoglobin concentration and oxygen saturation should be sustained with a judicious evidence-based transfusion strategy, inspired supplemental oxygen, and positive end-expiratory pressure. Lactate concentration, a surrogate marker of energy delivery to tissues and substrate utilization, should be followed at frequent intervals until shock has resolved. Maintaining the glucose level below 200 mg/dL may help mitigate any ongoing fluid losses due to osmotic diuresis. Glucose levels must be measured hourly when insulin therapy is used. Hyperglycemia can result in elevation of lactate levels.

Enteral nutrition should be delayed until the shock state has resolved and the patient is no longer receiving vasopressors. A nasogastric/orogastric tube should be placed to gravity drainage unless there is a specific indication for continuous or intermittent suction, such as abdominal distention. Gastric protection with an H_2 blocker or proton pump inhibitor should be considered for every patient. If a child is oliguric or has evidence of renal dysfunction, pertinent adjustments to drug doses should be made based on creatinine clearance; the trough levels of potentially nephrotoxic agents, such as vancomycin and aminoglycosides, should be monitored closely.

Triage tools, flow sheets, order sets, and management protocols expedite care and facilitate communication among healthcare providers; these tools should be drafted for both previously healthy children and those whose comorbidities place them at risk for sepsis. Measures that ensure optimal

care—including fluid resuscitation, prompt antibiotic administration, and titrated vasoactive infusions to achieve normalization of vital signs and central venous saturations >70%—translate into significant decreases in patient morbidity and mortality rates.

VI. ADDITIONAL CONSIDERATIONS

A. Management of Neonates and Young Infants

Infants with pulmonary and systemic circulations that are transitioning from the fetal to adult pattern are at risk for "persistent fetal circulation" and rapidly developing persistent pulmonary hypertension of the newborn. This culminates in acute right ventricular failure, manifested as shock with hepatomegaly and other indicators of worsening cardiac output. Myocardial dysfunction, ventricular dilatation, septal bowing and tricuspid regurgitation may be found on echocardiography.

In these infants, patency of the ductus arteriosus may result in pulmonary edema during volume resuscitation, mandating careful serial reevaluation and the use of discrete boluses of isotonic fluids. Titration of these fluids and vasoactive infusions reverses abnormalities in heart rate, capillary refill, and blood pressure, which may be difficult to monitor in facilities not accustomed to caring for such young patients.

While the cause of shock in the older child is generally easy to discover, the differential diagnosis is more diverse than in neonates. One of the most important causes is congenital heart disease, specifically ductus-dependent left-sided obstructive lesions that may have gone undetected until ductal closure and the onset of obvious shock. Any clinical suspicion of congenital heart disease should be addressed with appropriate support of ventilation and circulation, careful expansion of the intravascular space with isotonic fluids, and administration of inotropic and vasoactive infusions.

In neonates presenting with shock, an infusion of prostaglandin E_1 (0.05-0.1 μg/kg/min) must be started immediately and should be continued with close attention to possible adverse effects, including apnea, hypotension, and fever. The infusion should be continued until echocardiography excludes a lesion, such as coarctation of the aorta, interrupted aortic arch, critical aortic stenosis, or the constellation of malformations known as *hypoplastic left heart syndrome*.

Inborn errors of metabolism and congenital adrenal hyperplasia may present with hypoglycemia, metabolic acidosis, and evidence of end-organ dysfunction. These abnormalities must be recognized and treated early.

Appropriate management before PICU admission includes hourly monitoring of glucose and frequent assessments of ionized calcium and other electrolytes, in addition to other general supportive measures. Preterm and term neonates and young infants easily become hypothermic. Corticosteroids and other hormonal therapies (thyroid hormone) may play a role in the management of these patients, so close consultation is indicated.

Antibiotics must be initiated immediately, as a variety of pathogens may be rapidly fatal in neonatal sepsis.

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B. Shock in the Injured Child

A careful secondary trauma survey is essential as soon as fluid resuscitation has been initiated and the airway has been secured. Scalp lacerations may cause hypovolemia in patients of all sizes. Infants and toddlers with compliant skulls may develop hypovolemic shock from intracranial hemorrhage. Computed tomography scanning of suspected sites of injury (head, chest, abdomen, and pelvis) complement the initial basic trauma imaging obtained in the emergency department. Ultrasonography may be helpful if an experienced provider is available. Pelvic fractures may cause profuse internal hemorrhage and so must be stabilized mechanically using a sheet or appropriate device. Embolization of lacerated pelvic vessels may be lifesaving. Administration of packed red blood cells, including O-negative blood that has not been cross-matched, may also be lifesaving, especially in the presence of ongoing blood losses.

C. Adrenal Insufficiency

Young infants are at increased risk of adrenal insufficiency, particularly if they have hypoglycemia, hyponatremia, and hyperkalemia on their initial laboratory screen. Also at risk are: previously healthy children with profound shock refractory to conventional management; children with rapidly evolving rashes (especially if they are purpuric or petechial in nature), exposure to etomidate, a history of systemic steroid use within the last year, severe head or abdominal injury, or a history of panhypopituitarism; and those who have undergone cardiopulmonary bypass.

Patients with known or suspected adrenal insufficiency should be treated with hydrocortisone, which will provide both mineralocorticoid and glucocorticoid support at stress doses of at least 1 mg/kg as an IV bolus and 1 mg/kg (up to 50 mg) every 6 hours. Enteral fludrocortisone may be required in children with congenital adrenal hyperplasia.

D. Shock in Children with Diabetes

Children with new-onset diabetes presenting in diabetic ketoacidosis may have severe hypovolemia and hypoperfusion. Hemodynamic instability in these patients is appropriately addressed with volume resuscitation (10-20 mL/kg of normal saline or lactated Ringer solution). More fluid may be necessary in the presence of severely impaired organ perfusion. However, once hemodynamic stability has been attained, most intensivists and endocrinologists recommend repleting the intravascular space more conservatively than in other causes of hypovolemic shock, with fluid deficits usually corrected over 48 hours. Resuscitation fluids are commonly deducted from the first day's fluid dose, which is normally kept under 2500 to 3000 mL/m^2.

Gradual control of hyperglycemia is essential to correction of the catabolic state and ongoing fluid losses (**Chapter 8**). Low-dose (0.05-0.1 U/kg/h) regular insulin infusions are favored over larger doses, particularly during hospital transitions. Bedside glucose assays must be performed hourly to avoid excessive decreases, particularly when the child must be transported.

E. Refractory Shock

Shock that is refractory to initial treatment should raise suspicion of alternative diagnoses, such as congenital cardiac disease, acquired cardiac medical or trauma-related myocardial dysfunction, pericardial tamponade, pneumothorax, or hemothorax. Pulmonary hypertension, toxins, occult ongoing blood loss, inborn metabolic errors or adrenal insufficiency, abdominal trauma with evolving abdominal hypertension or compartment syndrome, and persistent infectious nidus may be challenging to diagnose. A high index of suspicion is key to the successful management of these entities.

F. Pediatric Shock in Resource-Rich Settings

Any delay—even a single hour—in fluid resuscitation, initiation of vasoactive infusions, or administration of antimicrobials causes significant increases in the risk of morbidity and mortality. Fluid resuscitation, mechanical ventilation, intensive monitoring, vasoactive infusions, and the use of extracorporeal therapies in concordance with the time-sensitive, goal-directed guidelines and institution-specific protocols has resulted in decreased morbidity and mortality rates. Implementation of recognition bundles to trigger rapid triage and identification, resuscitation and stabilization bundles to enhance adherence to best practices, and performance bundles to identify and overcome institution-specific barriers to implementation facilitates timely appropriate treatment.

G. Pediatric Shock in Resource-Limited Environments

Management of pediatric shock may be particularly challenging in non-industrialized, low-to-middle income, resource-limited environments. Malaria, measles, neonatal sepsis, and pneumonia are the leading causes of infection-related death in these settings. Simple preventive strategies have been shown to dramatically reduce morbidity and mortality rates. These strategies include access to clean water, promotion of breastfeeding, immunization, vitamin and mineral supplementation, timely administration of antibiotics, appropriate fluid resuscitation, and optimal perioperative care.

Educational strategies targeted at the lay public and healthcare providers may increase awareness, timely intervention, and outcomes. Context-specific “home-grown” trigger tools, protocols, and performance metrics can be designed and implemented using available paper-based tools. Specific protocols and procedures may be perfected with simulations using available technology.

Children at high risk for death appear to be those with preexisting or comorbid diseases, especially human immunodeficiency virus infection, malnutrition, or hypoxemia, or those exhibiting decreased mentation at presentation. The importance of early use of enteral rehydration (avoiding centrally acting antiemetics, such as promethazine) or parenteral fluid therapy via intraosseous or peripheral access cannot be overemphasized. Antibiotics should be administered as soon as possible.

Children known to have severe anemia appear to benefit from early blood transfusion rather than vigorous crystalloid or colloid infusion. The refeeding syndrome may occur in severely malnourished children. Frequent reassessment using simple clinical tools may help prevent pulmonary edema and other manifestations of hypervolemia in children with myocardial dysfunction. These children may benefit from cautious diuresis or appropriate vasoactive infusions, when available.

H. Pediatric Shock in the Context of Known Life-Limiting Conditions

Children with life-limiting conditions may present with shock. The initial approach to care when prognostic certainty or goals of care are unclear should mirror the management of otherwise healthy children while information is obtained. Careful consideration must be given to the overall goals of care. After close consultation with the primary physician and family members, invasive interventions, such as renal replacement therapy, and other modes of extracorporeal support known to improve outcomes in otherwise healthy children may be appropriately withheld in terminally ill children, with care redirected towards palliative goals.

Key Points

Diagnosis and Management of Shock

- Comprehensive initial and serial assessment of airway patency, breathing, and circulation are essential for the successful management of any seriously ill or injured patient.
- A point-of-care glucose assay and comprehensive laboratory panel should be performed.
- Oxygen should be administered to all patients at risk for shock, regardless of their initial pulse oximetry saturation readings.
- Vascular access should be secured within the first few minutes of presentation, with prompt consideration of intraosseous needle placement.
- Fluid resuscitation should begin with isotonic crystalloid, such as 0.9% normal saline or lactated Ringer solution, given in rapid boluses titrated to clinical response.
- Ketamine and atropine are the drugs of choice for airway control in the presence of shock.
- Antibiotics must be given early and certainly within an hour of presentation.
- Careful physical examination will reveal clues to the underlying cause of shock.
- Hypotension and bradycardia are a late findings in pediatric shock of any etiology and may precede full arrest.
- Vasoactive drugs should be considered early and selected based on the desired effect.
- In neonates or infants up to 2 months of age who have shock, consider ductus-dependent

congenital heart disease, hypovolemic shock, neonatal sepsis, inborn errors of metabolism, and congenital adrenal hyperplasia.

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ACUTE INFECTIONS

Objectives

- Recognize severe, life-threatening infections in children.
- Initiate timely, goal-directed management.
- Identify possible pathogens or pathology (including noninfectious) based on clinical findings and epidemiology.
- Select adequate initial antimicrobial treatment.
- Recognize and manage severe infections endemic in developing countries.

Case Study

An 11-year-old girl is admitted with a 2-day history of fever, abdominal pain, vomiting, and irritability. On physical exam, she is tachycardic (154 beats/min) and tachypneic (28 breaths/min); her abdomen is tender with guarding, rebound tenderness, and rigidity. Her white blood cell (WBC) count is 18,500/mm³. You suspect she is septic and initiate aggressive fluid resuscitation and antibiotic therapy.

Detection

– What physiologic parameters concern you about this patient?

– What site of infection would you suspect, and how would you investigate it?

– What diagnoses should be considered?

Intervention

– What additional tests are needed to confirm the diagnosis?

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

I. INTRODUCTION

Acute infections are common in the pediatric population and are often a component of acute illness. These infections may present with life-threatening symptoms, or they may have a subtle appearance and progress to severe conditions if undetected. Despite advances in the management of critically ill children, morbidity and mortality rates remain high in children with severe infections. This is related, in part, to the increase in high-risk pediatric populations encountered in clinical care, such as children with chronic conditions (eg, congenital heart disease, chronic pulmonary disorders) and immunosuppression (eg, congenital immune deficiencies, malignancies, and organ or stem cell transplants).

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Early recognition and management of severe infections prevents life-threatening complications.

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The outcome of acute infections in pediatric patients is directly related to the timely recognition of infections and the prompt initiation of appropriate therapy. Delayed diagnosis may result in significant organ dysfunction, shock, multiple organ failure, and death. Because the signs and symptoms of serious infections may be subtle, healthcare providers caring for these children must have a high index of suspicion. Close monitoring and frequent, thorough physical examination are important due to the dynamic process of disease progression. Age-dependent variations in vital signs must be recognized to detect physiologic derangements associated with acute infections (**Table 7-1**). Definitions of terms related to pediatric sepsis are presented in **Tables 7-2** and **7-3**.

Table 7-1	AGE-SPECIFIC UPPER VALUES (95TH PERCENTILE) FOR HEART RATE AND RESPIRATORY RATE, AND LOWER VALUES (5TH PERCENTILE) FOR SYSTOLIC BLOOD PRESSURE AND HEART RATE

Age Group	Tachycardia (beats/min)	Bradycardia (beats/min)	Tachypnea (breaths/min)	Leukocyte Count (x 10^3/mm^3)	Systolic Blood Pressure (mm Hg)
0 days-1 week	>180	<100	>50	>34	<65
1 week-1 month	>180	<100	>40	>19.5 or <5	<75
1 month-1 year	>180	<90	>34	>17.5 or <5	<100
2-5 years	>140	NA	>22	>15.5 or <6	<94
6-12 years	>130	NA	>18	>13.5 or <4.5	<105
13 to <18 years	>110	NA	>14	>11 or <4.5	<117

NA, not applicable

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Table 7-2 CRITERIA FOR SYSTEMIC INFLAMMATORY RESPONSE SYNDROME, INFECTION, SEPSIS, SEVERE SEPSIS, AND SEPTIC SHOCK

Systemic Inflammatory Response Syndrome

SIRS is characterized by the presence of at least 2 of the following 4 criteria, 1 of which must be abnormal temperature or abnormal leukocyte count:

- Core temperature (measured by rectal, bladder, oral, or central catheter probe) >38.5°C (>101.3°F) or <36°C (<96.8°F).
- Tachycardia, defined as a mean heart rate >2 SD above the normal for age in the absence of external stimulus, chronic drug use, or painful stimuli, or otherwise unexplained persistent elevation over a 30-min to 4-h period OR for children <1 year, bradycardia, defined as a mean heart rate <10th percentile for age in the absence of external vagal stimulus, β-blocker drugs, or congenital heart disease, or otherwise unexplained persistent depression over a 30-min period.
- Mean respiratory rate >2 SD above normal for age or mechanical ventilation for an acute process unrelated to underlying neuromuscular disease or the receipt of general anesthesia.
- Leukocyte count elevated or depressed for the patient's age (not secondary to chemotherapy-induced leukopenia) or >10% immature neutrophils.

Infection

Infection is proven by a culture, tissue stain, or nucleic acid detection (eg, polymerase chain reaction test) of a pathogen. A suspected infection is a clinical syndrome without detection of a microorganism associated with a high probability of infection. Evidence of infection may include positive findings on clinical examination (eg, petechial or purpuric rash, or purpura fulminans), imaging (eg, free air in the abdomen indicating a perforated viscus or a chest radiograph consistent with pneumonia), or laboratory tests (eg, elevated concentration of white blood cells in a normally sterile body fluid).

Sepsis

SIRS in the presence of, or as a result of, suspected or proven infection.

Severe Sepsis

Sepsis with cardiovascular organ dysfunction before treatment or corrected by fluid resuscitation of <40 mL/kg/h in <1 h OR acute respiratory distress syndrome OR dysfunction of ≥2 other organs. Organ dysfunction is defined in **Table 7-3**.

Septic Shock

Sepsis with cardiovascular organ dysfunction as defined in **Table 7-3**.

SIRS, systemic inflammatory response syndrome; SD, standard deviation

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Table 7-3 ORGAN DYSFUNCTION CRITERIA

Cardiovascular Dysfunction

Despite administration of isotonic fluid bolus ≥40 mL/kg in 1 h:

- Decrease in BP (hypotension) <5th percentile for age or systolic BP <2 SD below normal for age

  OR

- Need for vasoactive drugs to maintain BP in normal range

  OR

- 2 of the following:
  - Unexplained metabolic acidosis: base deficit >5 mmol/L
  - Increased arterial lactate >2 times upper limit of normal
  - Oliguria: urine output <0.5 mL/kg/h
  - Prolonged capillary refill: >5 s
  - Core to peripheral temperature gap >3ºC (>37.4ºF)

Respiratory[a]

- Pao_2/F_{IO_2} <300 in absence of cyanotic heart disease or preexisting lung disease

  OR

- $Paco_2$ >20 mm Hg over baseline reading

  OR

- Proven need for >50% F_{IO_2} to maintain saturation ≥92%

  OR

- Need for nonelective invasive or noninvasive mechanical ventilation

Neurologic Dysfunction

- Glasgow Coma Scale score ≤11

  OR

- Acute change in mental status with a decrease in Glasgow Coma Scale score ≥3 points from abnormal baseline

Hematologic Dysfunction

- Platelet count <80,000/mm³ or a decline of 50% in platelet count from highest value recorded over the past 3 days (for long-term hematology/oncology patients)

  OR

- International normalized ratio >2

Renal Dysfunction

- Serum creatinine ≥2 times upper limit of normal for age or a 2-fold increase in baseline creatinine

Hepatic Dysfunction

- Total bilirubin ≥4 mg/dL (not applicable for newborn)

  OR

- ALT 2 times upper limit of normal for age

BP, blood pressure; SD, standard deviation; F_{IO_2}, fraction of inspired oxygen; ALT, alanine transaminase

[a]Acute respiratory distress syndrome must include a Pao_2/F_{IO_2} ratio ≤200 mm Hg, bilateral infiltrates, acute onset, and no evidence of left heart failure. Acute lung injury is defined identically, except the Pao_2/F_{IO_2} ratio must be ≤300 mm Hg. Proven need assumes oxygen requirement was tested by decreasing flow, subsequently increasing flow if required. In postoperative patients, this requirement can be met if the patient has developed an acute inflammatory or infectious lung process that prevents extubation.

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II. DIAGNOSIS

Illnesses caused by infections have a wide variety of signs and symptoms, some of which are clear and easy to detect, while others are subtle and nonspecific. A thorough and meticulous physical exam, and an understanding of age-related differences in anatomy and physiology, are vitally important in identifying an infectious cause of illness. A high index of suspicion for an infectious etiology should always be maintained. The earlier a severe infection is diagnosed, the better the outcome the patient generally will have. Prompt recognition of a severe infection is based on a clear understanding of the risk factors, epidemiology, and the findings on the physical exam. The diagnostic process can also be aided by laboratory and radiologic examinations. Properly performed microbiologic testing can be an invaluable tool to making the correct diagnosis.

A. General Signs of Infection

The host responds to an invading microorganism with a cascade of immune-mediated events. These lead to the activation of numerous physiologic systems, resulting in responses that should alert the clinician to the possibility of an infection.

Fever is the most common sign of infection. The best estimate of the core temperature is taken rectally, but an oral or bladder temperature reading is acceptable. A core temperature above 38°C (100.4°F) is usually considered a fever, but a temperature of 38.5°C (101.3°F) improves the specificity and is a commonly used threshold in clinical practice. Patients who are immunocompromised have a significantly higher risk of acquiring life-threatening infections, and a temperature of 38°C (100.4°F) sustained over a 1-hour period or a single oral temperature measurement of 38.3°C (101°F) should lead to further investigation. Most infections in children present with fever; however, some patients, particularly neonates, with severe infections may present with hypothermia (temperature <36°C [<96.8°F]). Fever may be caused by a wide variety of noninfectious etiologies as well, such as inflammatory diseases, drugs, blood products, neoplasms, endocrine disorders, central nervous system bleeding, thrombosis, recent surgery, or iatrogenic causes, such as excessive patient bundling. Temperatures >41°C (>105.8°F) are seldom associated with an infectious cause.

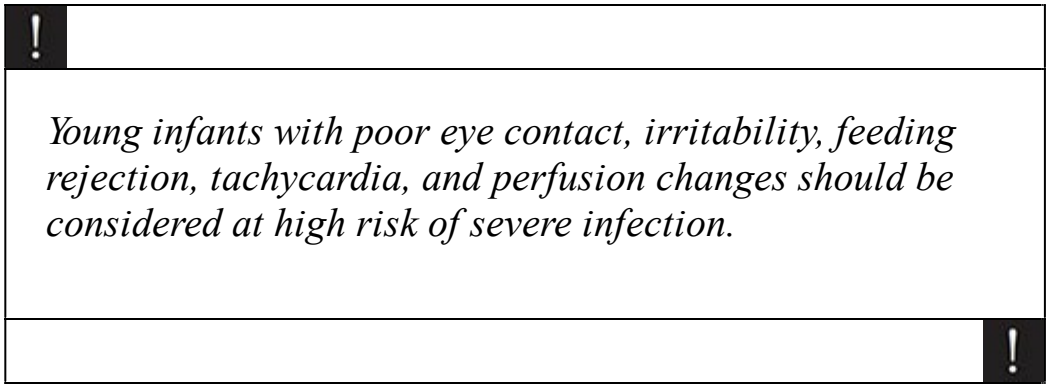
!
Young infants with poor eye contact, irritability, feeding rejection, tachycardia, and perfusion changes should be considered at high risk of severe infection.
!

Table 7-4 outlines signs of infection specific to organ systems. Signs may indicate a specific site of infection or reflect the systemic effect of a remote infection.

Table 7-4 GENERAL SYMPTOMS AND SIGNS OF INFECTION BASED ON ORGAN SYSTEM INVOLVED

Organ System	Clinical Finding
Cardiovascular	Tachycardia Diminished or bounding peripheral pulses Gallop rhythm Murmur or rub
Respiratory	Tachypnea Cough Dyspnea Chest pain Nasal flaring Grunting Retractions Cyanosis *Auscultation*: crackles, decreased breath sounds, abnormal vocal resonance
Central nervous system	*Infants*: fever, lethargy, irritability, bulging anterior fontanelle, apnea, seizure, poor eye contact *Children*: fever, headache, vomiting, irritability, mental status changes, nuchal rigidity, focal neurologic signs
Urinary tract infection	*Infants*: fever, irritability, vomiting, poor feeding, hyperbilirubinemia, failure to thrive *Children*: dysuria, urgency, increased frequency of voiding
Cutaneous/soft tissue infection	Pain Erythema Induration Warmth +/- fever Systemic involvement possible
Abdominal infection	Fever Diffuse abdominal pain Vomiting Tenderness +/- rebound tenderness Rigid abdominal wall
Septic arthritis or osteomyelitis	Fever Pain Swelling Erythema Tenderness Limited movement
Nonspecific	*Children:* chills, myalgia, fatigue, anorexia *Neonates:* irritability, poor feeding, mottled skin

Skin manifestations (rashes) are common in the clinical presentation of a variety of infectious organisms. A basic understanding of the types of skin manifestations is essential to making an accurate assessment and determining the severity and acuteness of the patient's illness. Common skin lesions are given in **Table 7-5**. Maculopapular rashes are most commonly seen in viral illnesses, immune-mediated syndromes, and some bacterial infections. Pustules most commonly are due to a bacterial infection. Vesicles are commonly caused by herpes simplex or varicella virus; impetigo due to *Staphylococcus aureus* or *Streptococcus pyogenes* may result in bullae or vesicles. The location of the rash on the body (ie, peripheral or central distribution) may be helpful in narrowing the differential diagnosis. Petechial rashes deserve immediate attention and evaluation to rule out severe

illness. Petechiae with a distribution limited to below the umbilicus are more highly associated with a severe infection.

Table 7-5	COMMON PRIMARY SKIN LESIONS
Lesion Type	**Description**
Macule	Circumscribed area of change in normal skin color, with no skin elevation or depression; may be any size
Papule	Solid, raised lesion <0.5 cm in greatest diameter
Nodule	Similar to papule but located deeper in dermis or subcutaneous tissue; differentiated from papule by palpability and depth, rather than size
Plaque	Elevation of skin occupying a relatively large area in relation to height; often formed by confluence of papules
Pustule	Circumscribed elevation of skin containing purulent fluid of variable character (ie, fluid may be white, yellow, greenish, or hemorrhagic)
Vesicle	Circumscribed, elevated, fluid-containing lesion <0.5 cm in greatest diameter; may be intraepidermal or subepidermal in origin
Bulla	Same as vesicle, except lesion >0.5 cm in greatest diameter

Classified using Fitzpatrick TB, Johnson RA, Polano MK, et al. *Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases*. 3rd ed. New York, NY: McGraw-Hill; 1997. Habif TP, ed. *Clinical Dermatology: A Color Guide to Diagnosis and Therapy*. 3rd ed. St. Louis, MO: Mosby; 1996.

B. Laboratory Findings

A complete blood count with differential count is routinely performed in a patient suspected of having a severe infection. Leukocytosis (WBC count >12,000/mm^3) may be a reaction to an infectious or an inflammatory process, among other causes. Most bacterial infections cause leukocytosis with neutrophilia (neutrophil count ≥8,000/mm^3 in adults and children older than 1 year); however, severe infections may present with leukopenia (leukocyte count <4,000/mm^3), especially in young infants. The presence of leukocytosis or neutrophilia alone cannot accurately predict the presence or absence of bacterial infection. Platelets are acute phase reactants, so thrombocytosis (platelet count >450,000/mm^3) can be an indirect sign of infection; however, thrombocytopenia (platelet count <150,000/mm^3) and other clotting abnormalities are well-known complications of sepsis.

Alterations in the prothrombin time and activated partial thromboplastin time can be suggestive of disseminated intravascular coagulation during severe sepsis. Elevated D-dimers and fibrin degradation products in association with low fibrinogen confirm the diagnosis, although other clinical entities, such as thrombotic thrombocytopenic purpura, must be excluded. Acute phase reactant biomarkers that may indicate the presence of an acute infection, although not specific, are an elevation of C-reactive protein, erythrocyte sedimentation rate, and procalcitonin. In the acute setting, when used together with other markers of inflammation and with clinical criteria, these may help in identifying the presence of an infectious etiology. Trending the levels of these biomarkers may also aid in determining the effectiveness of treatment.

To assess the adequacy of oxygen delivery, a blood gas measurement (arterial or capillary) is useful. A metabolic acidosis accompanied by high lactate levels or low bicarbonate levels indicates an

imbalance in the delivery of oxygen compared to tissue demand. Differences in arterial and central venous oxygen concentrations can also assess the adequacy of oxygenation, with a difference of more than 25% to 30% indicating inadequate delivery. The Pao_2 or saturation on pulse oximetry (Spo_2) may be used to confirm oxygenation and help in the diagnosis of conditions such as acute respiratory distress syndrome. The $Paco_2$ can be low during respiratory compensation (<35 mm Hg [4.6 kPa]) or elevated (>45 mm Hg [5.9 kPa]) during a disease with ventilation impairment. Also, co-oximetry measurement of mixed venous oxygen saturation using blood samples from a central venous catheter (preferably from the superior vena cava) is useful in guiding treatment during severe sepsis (**Chapter 6**).

Inadequate oxygen delivery may also be recognized clinically. In particular, alterations in central nervous system and renal functions are early signs of impending cardiovascular compromise. The brain and kidneys receive 20% of cardiac output; if compromised by the effect of a severe infection, alterations in organ function occur. These alterations may be subtle (eg, agitation, anxiety, lethargy, somnolence) or be more profound (eg, encephalopathy). Conversely, normal urine output and mental status generally suggest adequate cardiac output. Severe infections may affect renal function directly (by nephrotoxins, as in the hemolysis of malaria) or indirectly (by decreased renal blood flow, by shock); therefore, blood urea nitrogen and serum creatinine should always be measured. Newer serum and urine biomarkers, such as neutrophil gelatinase-associated lipocalin, are emerging as early indicators of kidney injury. During acute kidney injury, doses of renally excreted drugs (eg, many antibiotics) should be adjusted to creatinine clearance.

Hypoglycemia (in neonates and young infants) or hyperglycemia may be present during severe infections. Hyperglycemia has been associated with increased mortality in critically ill adults, and the use of insulin to achieve normoglycemia (while assiduously avoiding hypoglycemia) may improve mortality and morbidity rates in surgical ICUs. The use of insulin therapy is controversial in pediatric patients; however, hyperglycemia is commonly treated in children when glucose values are persistently above 180 to 200 mg/dL (9.98-11.1 mmol/L). Data also suggest that large variations in blood glucose levels may be associated with poor outcomes. Blood glucose should be routinely measured at the bedside as part of the initial stabilization and then followed through the course of illness. In recent studies, maintaining strict euglycemia with insulin therapy did not show benefit in pediatric patients undergoing cardiopulmonary bypass for correction of congenital heart lesions or in children with cardiovascular or respiratory failure; such treatment may impose a greater risk of harm,

Lastly, as all organ systems can be primarily or secondarily affected by an acute infection, measures of serum electrolytes (sodium, potassium, calcium, phosphorus, magnesium, or serum bicarbonate) and liver enzyme and renal function tests need to be followed (**Chapter 8**). Further, various infectious syndromes are defined by derangements in certain of these laboratory values.

C. Microbiology Tests

Microbiology tests are essential in establishing the microbial etiology. Healthcare providers must order the correct laboratory tests and submit the correct specimens to improve the diagnostic sensitivity and specificity.

Sterilely collected specimens of any body fluid, infected tissue, and blood should be obtained for culture. Gram stain can rapidly provide important information that can be applied immediately to patient care. It is particularly useful in the diagnostic confirmation of bacterial meningitis, urinary tract infection, or other infected fluid collections, such as abscesses, empyema, and peritonitis. Specimens obtained via biopsy or bronchoscopy should be sent routinely for Gram stain. Other stains should be considered (eg, acid-fast bacilli, silver stain [pneumocystis], fungal), depending on the type of infection that is suspected. The growth of microorganisms in specific growth media (aerobic, anaerobic, fungal, mycobacterial) establishes the microbiologic diagnosis and can allow testing for antimicrobial susceptibility. Any material obtained appropriately can be sent for culture. A minimum of 2 mL of blood should be drawn whenever possible for optimizing the chances of isolating a blood pathogen. For patients ≥45 kg, a 2-bottle aerobic and anaerobic adult blood culture system should be inoculated with 8 to 10 mL per bottle. If possible, one set of blood cultures should be taken from ≥2 separate sites (ie, ≥2 sets) to increase specificity. Malaria is still routinely identified on blood films: a thick film to identify parasites and a thin film to determine the species. Also available are rapid diagnostic tests that provide quick, point-of-care information about the involved species. Antigen-detection tests using direct immunofluorescence, enzyme immunoassay, or latex agglutination techniques for rapid detection of bacterial antigens can be performed using a variety of specimens, such as urine, sputum, or throat swab. Antigen detection tests can give useful information; however, because the sensitivity of that information can be low, initial antibiotic management of severe infections should not be based solely on the results of these tests.

Polymerase chain reaction (PCR) for the detection of microbial DNA or RNA can be a useful technique for the detection of diverse microorganisms. It is the standard and preferred methodology to detect herpes viruses and enterovirus in cerebrospinal fluid (CSF).

Serologic testing for the detection of antibodies is the test of choice for some infections, such as Epstein-Barr virus infection, Rocky Mountain spotted fever, and dengue, and for some fungal infections.

III. INFECTIOUS SYNDROMES

A. Sepsis

Sepsis is the result of activation of the systemic inflammatory response syndrome (**Table 7-2**) caused by an identified or presumed infection. This syndrome can have a high mortality rate, particularly if not treated adequately or in a timely fashion. The use of intensive care and goal-directed therapies has been shown to improve outcome.

!

Rapid initiation of adequate empirical antibiotic therapy is imperative for lowering the mortality rate during severe infection. Local resistance patterns must be considered in

the choice of empiric antibiotics.

The clinical definition of systemic inflammatory response syndrome (and therefore sepsis) in children differs slightly from the adult definition. In children, temperature or leukocyte abnormalities must be present, in addition to abnormalities of the heart rate or respiratory rate. Bradycardia may be a sign of the syndrome in the newborn age group, but not in older children (in whom it is a near-terminal event).

Hypotension is a late manifestation in children with septic shock.

Early clinical suspicion should be based on the presence of hyperthermia or hypothermia, changes in mental status, and alterations in peripheral perfusion. Evaluation of children with suspected sepsis is based on a meticulous and repeated physical assessment. Heart rate, capillary refill, respiratory rate, mental status, urinary output, and alteration in the skin's appearance should be evaluated carefully.

Management should be initiated as soon as sepsis is suspected. The goal of therapy is to prevent or correct abnormalities that arise due to inadequate oxygen delivery at the cellular level. This means maintaining adequate oxygenation of hemoglobin, adequate hemoglobin levels, and sufficient cardiac output to deliver oxygen where it is needed.

As with all critically ill patients, the main priorities are assessment of the airway, proper ventilation, establishment of adequate circulation, and prompt administration of appropriate antibiotics. Fluid and inotropic management of the septic patient is discussed at length in **Chapter 6**.

Early recognition of the septic patient and prompt, aggressive therapy are crucial for an optimal outcome. After cultures have been obtained, administering appropriate antibiotics within the first hour of disease recognition is recommended. If appropriate cultures cannot be obtained in a timely manner, initiation of broad-spectrum empiric antibiotics should not be delayed. Goal-directed initial fluid resuscitation is also fundamental in managing these patients. In resource-rich settings (with access to inotropes, vasopressors, and mechanical ventilation) and after rapidly obtaining IV or intraosseous access, the resuscitation should begin with 20 mL/kg isotonic crystalloid boluses administered over 5 to 10 minutes. Patients usually require between 40 and 60 mL/kg, but higher volumes may be needed. Fluid resuscitation should be targeted to improvement in physiologic parameters: normalization of heart rate, urine output >1 mL/kg/h, capillary refill <2 seconds, and normal mental status. Blood pressure alone is not a reliable end-point for assessing the adequacy of fluid resuscitation (**Chapters 6** and **8**). Hepatomegaly occurs in fluid overload; therefore, when it develops, it indicates adequacy of fluid resuscitation.

If hemodynamic goals have not been achieved despite appropriate fluid resuscitation, vasopressors or inotropes should be given. Dopamine is recommended as the first medication, but 2016 data suggest that epinephrine may be a superior first-line agent. In cases of dopamine-resistant shock, a second agent must be added. The choice of medications should be determined by the clinical exam: direct vasopressors or vasodilators may be needed in the presence of low cardiac output with low or high systemic vascular resistance, respectively.

Therapeutic goals for patients in septic shock include improvements in the clinical findings noted previously, as well as other measures of oxygen delivery. Targeting a decrease in serum lactate or central venous oxygen saturation >70% can aid in patient management and lead to improved outcomes (**Figure 7-1**).

Figure 7-1. Approach to Pediatric Shock

0 min

Recognize decreased mental status and perfusion.
Begin high flow O_2 and establish IO/IV access according to PALS.

5 min

If no hepatomegaly or rales / crackles then push 20 mL/kg isotonic saline boluses and reassess after each bolus up to 60 mL/kg until improved perfusion. Stop for rales, crackles or hepatomegaly. Correct hypoglycemia and hypocalcemia.
Begin antibiotics.

15 min

Fluid refractory shock?

Begin peripheral IV/IO inotrope infusion, preferably Epinephrine 0.05 – 0.3 µg/kg/min
Use Atropine / Ketamine IV/IO/IM if needed for Central Vein or Airway Access

Titrate Epinephrine 0.05 – 0.3 µg/kg/min for Cold Shock.
(Titrate central Dopamine 5 – 9 µg/kg/min if Epinephrine not available)
Titrate central Norepinephrine from 0.05 µg/kg/min and upward to reverse Warm Shock.
(Titrate Central Dopamine ≥ 10 µg/kg/min if Norepinephrine not available)

60 min

Catecholamine-resistant shock?

If at risk for Absolute Adrenal Insufficiency consider Hydrocortisone.
Use Doppler US, PICCO, FATD or PAC to Direct Fluid, Inotrope, Vasopressor, Vasodilators
Goal is normal MAP-CVP, $ScvO_2$ > 70%* and CI 3.3 – 6.0 L/min/m²

Normal Blood Pressure Cold Shock	Low Blood Pressure Cold Shock	Low Blood Pressure Warm Shock
$ScvO_2$ < 70%* / Hgb > 10g/dL on Epinephrine?	$ScvO_2$ < 70%* / Hgb > 10g/dL on Epinephrine?	$ScvO_2$ > 70%* on Norepinephrine?
Begin Milrinone infusion. Add Nitroso-vasodilator if CI < 3.3L/min/m² with High SVRI and/or poor skin perfusion. Consider Levosimendan if unsuccessful.	Add Norepinephrine to Epinephrine to attain normal diastolic blood pressure. If CI < 3.3 L/min/m² add Dobutamine, Enoximone, Levosimendan, or Milrinone.	If euvolemic, add Vasopressin, Terlipressin, or Angiotensin. But, if CI decreases below 3.3 L/min/m² add Epinephrine, Dobutamine, Enoximone, Levosimendan.

Persistent Catecholamine-resistant shock?

Evaluate Pericardial Effusion or Pneumothorax,
Maintain IAP < 12mmHg

Refractory Shock?

ECMO

IO, intraosseous; IM, intramuscular; MAP-CVP, mean arterial pressure-central venous pressure; $Scvo_2$, central venous oxygen saturation; Hgb, hemoglobin; PICCO, pulse-induced contour cardiac output; FATD, femoral arterial thermodilution technique; ECMO, extracorporeal membrane oxygenation; SVRI, systemic vascular resistance index

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1. Steroids

There is continuous debate regarding the use of steroids in pediatric sepsis. Current guidelines recommend the use of hydrocortisone in the presence of catecholamine-resistant shock and risk of adrenal insufficiency. Risk factors for adrenal insufficiency include meningococcemia, long-term use of steroids, and pituitary or adrenal abnormalities. Recommended initial doses of hydrocortisone may vary from 50 mg/m²/d for stress coverage to 50 mg/kg/d as a 24-hour infusion titrated for reversal of refractory shock. When possible, blood cortisol concentrations should be obtained before the first dose of hydrocortisone.

2. Antibiotic Therapy

Antibiotics should be administered within the first hour of treatment, immediately after cultures have been obtained. However, if microbiology samples are difficult to obtain, treatment should be started without delay.

Broad-spectrum antibiotics should be given empirically with the choice of agent dependent on the patient's clinical, laboratory, and imaging characteristics. Considerations vary with the patient's age, physical findings, immunological status, previous hospitalizations, previous antibiotic treatment, and presence of central venous catheters or other medical devices. Knowledge of local microbe resistance patterns may also help guide the antimicrobial selection. Possible sepsis pathogens are summarized in **Table 7-6**.

Table 7-6 POSSIBLE SEPSIS PATHOGENS

Age Group	Pathogens
0-30 days	Group B streptococci, *Escherichia coli* and other Enterobacteriaceae, *Staphylococcus aureus*, and *Listeria monocytogenes*
1-3 months	*Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae*, and *E coli* and other Enterobacteriaceae
3 months-5 years	*S pneumoniae, N meningitidis, H influenzae*, and *S aureus*
>5 years	*S pneumoniae, N meningitidis*, group Aβ-hemolytic streptococcus, *S aureus*, and *Fusobacterium necrophorum*
Immunocompromised or hospitalized children	*S aureus, Staphylococcus epidermidis* and other coagulase-negative staphylococci, *Streptococcus mitis*, Gram-negative bacilli, and *Candida* Other considerations: *Salmonella*, leptospirosis, tularemia, typhus group, *Brucella*, babesiosis

Antibiotic treatment depends on the suspected infection. For community-acquired sepsis in immunocompetent patients, cefotaxime or ceftriaxone with vancomycin provides adequate initial empiric coverage in most nontropical areas. In certain areas of the world, organisms such as *Rickettsia, Burkholderia*, and malaria are prevalent, and antimicrobials directed at these organisms should also be considered as initial empiric therapy. In patients with risk factors such as neutropenia or in hospitalized children, Gram-negative coverage should be expanded with an aminoglycoside in

combination with an extended spectrum β-lactam (eg, piperacillin-tazobactam or meropenem). If a central venous catheter is present, vancomycin should be added. In addition, septic shock in a patient with an indwelling central venous catheter and no other focus of infection is an indication for prompt removal of the catheter, as soon as feasible. Empiric antibiotic choices for severe infections acquired outside of hospital settings are summarized in **Table 7-7**. Antibiotic doses are described in **Table 7-8**.

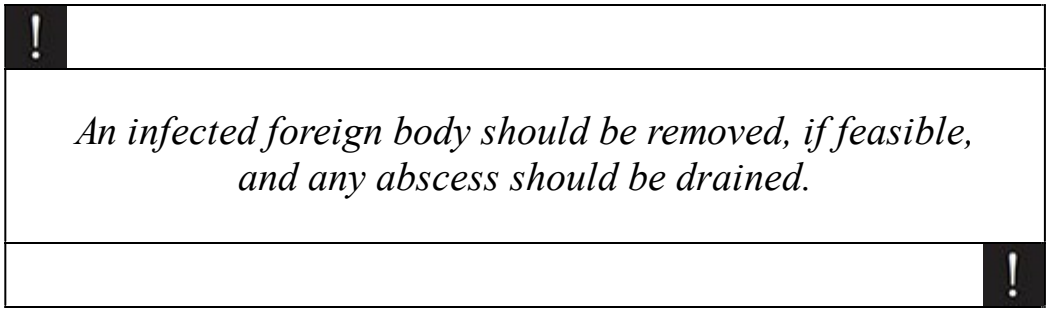


Table 7-7 EMPIRIC ANTIBIOTIC CHOICES FOR COMMUNITY-ACQUIRED SEPSIS[a]

Age	Suspected Bacteremia	Sepsis Syndrome and/or Severe Infection
<4 weeks	Ampicillin **AND** cefotaxime or gentamicin	Ampicillin **AND** vancomycin **AND** cefotaxime or gentamicin
4-7 weeks	Ampicillin **AND** ceftriaxone or cefotaxime	Ampicillin **AND** vancomycin **AND** ceftriaxone or cefotaxime
8 weeks-adolescence	Ceftriaxone or cefotaxime	Vancomycin **AND** clindamycin **AND** ceftriaxone or cefotaxime

[a]Local resistance patterns must be considered when selecting empiric antibiotics for community-acquired sepsis.

Table 7-8 ANTIMICROBIAL USE IN THE TREATMENT OF SEPSIS

Age	Group Treatment[a]
Neonates ≤7 days	• Ampicillin 50-100 mg/kg/dose every 12 h The higher doses are for meningitis or group B streptococcal sepsis. • Gentamicin – <29 wk: 5 mg/kg/dose every 48 h – 30-34 wk: 4.5 mg/kg/dose every 36 h – >35 wk: 4 mg/kg/dose every 24 h (depending on gestational age) • Cefotaxime 50 mg/kg/dose every 12 h
Neonates >7 days	• Ampicillin 50-75 mg/kg/dose every 6-8 h, depending on gestational age The higher doses are for meningitis or group B streptococcal sepsis.

	• Gentamicin 4 mg/kg/dose every 24 h (if >30 wk); interval depends on plasma levels • Cefotaxime 50 mg/kg/dose every 8-12 h, depending on gestational age • Vancomycin 15 mg/kg/dose every 6-12 h, depending on gestational age • Metronidazole 7.5 mg/kg/dose every 12-48 h, depending on gestational age
Previously healthy infants and children	• Ceftriaxone 50 mg/kg/dose every 24 h; if dose >2 g, divide and administer every 12 h • Vancomycin 15 mg/kg/dose every 6 h for CNS infection or 15 mg/kg/dose every 8 h for non-CNS infections • Metronidazole 7.5 mg/kg/dose every 6 h
Hospitalized or immunosuppressed children	• Ceftriaxone 75-100 mg/kg/day every 24 h; if dose >2 g, divide and administer every 12 h; use higher dose for meningitis. • Gentamicin 2-2.5 mg/kg/dose every 8 h • Amikacin 7.5-10 mg/kg/dose every 8 h • Ceftazidime 50 mg/kg/dose every 8 h • Metronidazole 7.5 mg/kg/dose every 6 h • Vancomycin 15 mg/kg/dose every 6 h for CNS infection or 15 mg/kg/dose every 8 h for non-CNS infections • Meropenem 20 mg/kg/dose every 8 h; 40 mg/kg/dose every 8 h for meningitis

CNS, central nervous system

[a]Local resistance patterns need to be considered when selecting empiric antibiotics for community-acquired sepsis.

3. Other Considerations

Useful blood tests during initial stabilization include blood gas, lactate, electrolytes, blood urea nitrogen, creatinine, glucose, complete blood count and differential count, and coagulation parameters (prothrombin time, activated partial thromboplastin time, fibrinogen, and d-dimer). Maintaining a hemoglobin level of at least 10 g/dL has been recommended to optimize oxygen delivery in a patient with clinical sepsis or in septic shock. Hypoglycemia and electrolyte abnormalities should be corrected. Although tight glycemic control has been advocated for many years, recent data suggest that the use of insulin to maintain a normal range of blood glucose levels is no longer warranted and, in fact, can lead to added morbidities.

Lastly, in patients with refractory septic shock and/or respiratory failure despite maximal conventional therapies, the use of extracorporeal membrane oxygenation can be considered.

B. Toxic Shock Syndrome

Case Study

A 16-year-old girl presents to the emergency department with a temperature of 40°C (104°F), malaise, headache, confusion, diarrhea, and skin rash. She is not sexually active and started her

menstrual period 2 days ago. Her pulse rate is 130 beats/min and blood pressure is 98/50 mm Hg. She has a diffuse erythroderma and mild abdominal tenderness. You are concerned about this patient's condition and immediately initiate therapy.

Detection

– What is the likely etiology of this illness?

– How would you describe her physiologic condition?

Intervention

– What therapy should be initiated?

Reassessment

– After fluid resuscitation, what other therapies should be considered?

Effective Communication

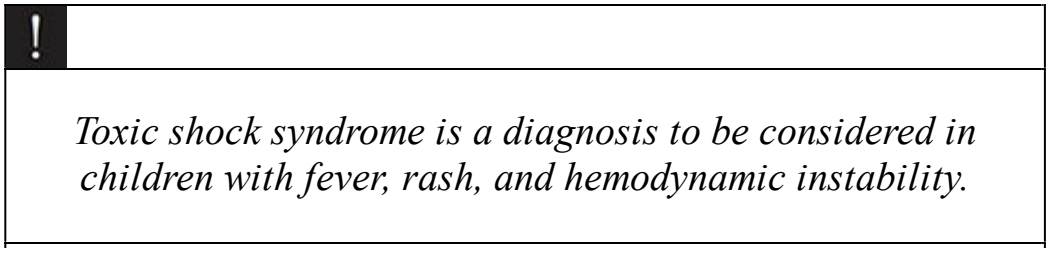
– When the patient's clinical status changes, who needs this information and how will it be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

Toxic shock syndrome (TSS) is a life-threatening syndrome that should be considered in ill patients with fever and erythroderma, with or without hypotension. The syndrome, caused by *S aureus* or *S pyogenes*, was first described in children with postoperative wounds, but an epidemic was recognized in young women in the 1980s due to use of highly absorbent tampons during menstruation. Since then, the incidence of TSS with tampons has sharply declined, but cases continue to occur in the absence of tampon use. Nonmenstrual TSS has been associated with *S aureus* infections in surgical and postpartum wounds, mastitis, sinusitis, respiratory illness (especially following influenza), osteomyelitis, arthritis, burns, and other skin and soft tissue infections. Most of the cases are due to methicillin-susceptible *S aureus*, but cases with methicillin-resistant strains have been reported. The TSS toxin, namely TSST-1 or staphylococcal enterotoxin B, acts as a superantigen and binds to and stimulates certain T cells independent of major histocompatibility complex presentation. Stimulated T cells release tumor necrosis factor and other chemokines that cause fever, rash, and capillary leakage, resulting in hypotension and organ damage.



Patients often present with acute onset of fever, sore throat, intense myalgia, profuse diarrhea, and sometimes vomiting. The rash in TSS, which is an early finding, is described as erythroderma and has sometimes been mistaken for a sunburn rash, which it closely resembles. A scarlatiniform rash can also been seen. Patients may present with listlessness or confusion. A nonpurulent conjunctival hyperemia, pharyngeal inflammation, or strawberry tongue can also be seen. TSS affects multiple organ systems, and patients can present with signs of acute respiratory distress syndrome, renal failure, or other signs of gastrointestinal and hematologic involvement. Progression to fulminant shock can occur rapidly. Desquamation of digits, palms, and soles occurs typically in convalescence.

The diagnostic criteria for TSS due to *S aureus* are shown in **Table 7-9**. Blood cultures are rarely positive as the organism need not spread systemically; this is an exotoxin-mediated syndrome. *S aureus* can be isolated from wound, respiratory, or other mucosal sites, although its isolation is not required for the diagnosis of staphylococcal TSST-1. Other abnormalities in laboratory results reflect the degree of organ involvement, such as transaminase elevation, coagulopathy, and elevated creatinine.

Table 7-9 DEFINITION OF TOXIC SHOCK SYNDROME DUE TO STAPHYLOCOCCUS AUREUS[a]

1. **Fever**
   Temperature >38.9°C (>102.0°F)
2. **Hypotension**
   Systolic blood pressure: ≤90 mm Hg for adults or <5th percentile by age for children <16 years of age
   Orthostatic drop in diastolic blood pressure ≥15 mm Hg
   Orthostatic syncope or dizziness
3. **Rash**
   Diffuse macular erythroderma
4. **Desquamation**
   1-2 weeks after onset of illness, particularly involving palms and soles
5. **Multisystem involvement (>3 of the following organ systems)**
   Gastrointestinal: Vomiting or diarrhea at onset of illness
   Muscular: Severe myalgia or creatine phosphokinase elevation >2 times normal upper limit
   Mucous membranes: Vaginal, oropharyngeal, or conjunctival hyperemia
   Renal: Blood urea nitrogen or serum creatinine >2 times normal upper limit, or pyuria (>5 white blood cells per high-powered field)
   Hepatic: Bilirubin or transaminases >2 times the normal upper limit
   Hematologic: Platelets <100,000/µL
   Central nervous system: Disorientation or alterations in consciousness without focal neurologic signs in the absence of fever and hypotension

Laboratory criteria: negative results on the following tests, if obtained
Blood, throat, or cerebrospinal fluid cultures for another pathogen (blood cultures may be positive for *S aureus*)
Serologic tests for Rocky Mountain spotted fever, leptospirosis, or measles

[a]Case definition of toxic shock syndrome due to *S aureus* from the Centers for Disease Control and Prevention.Requires meeting laboratory criteria plus 5 clinical findings including desquamation (unless the patient dies before desquamation occurs) for a confirmed case; meeting laboratory criteria plus 4 of 5 clinical findings for a probable case.

TSS can also be caused by *S pyogenes* (group A β-hemolytic streptococci [GABHS]) and is seen in children with focal infections, such as myositis, pneumonia, necrotizing fasciitis, or during varicella infection. The clinical features and lab abnormalities are similar to those caused by *S aureus*.

Patients with suspected TSS should receive clindamycin in addition to vancomycin. Nafcillin can also be started initially and continued or stopped once the susceptibility of *S aureus* is available. Surgical drainage of any identified collections should be undertaken, in addition to removal of any tampon present. Administration of IV immune globulin (IVIG) can be considered in TSS caused by *S aureus*, particularly in patients who are severely ill and have not responded to the initial supportive measures and antibiotics; however, no controlled data support the use of IVIG.

C. Meningococcemia

Case Study

A 15-year-old boy is admitted with sudden onset of high fever, shaking chills, vomiting, and diffuse non-blanching skin lesions. Vital signs show he is tachycardic and hypotensive.

Detection

– What physiologic parameters concern you about this patient?

– What is the most likely diagnosis? The worst possible diagnoses?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs the information and how will it be disseminated?

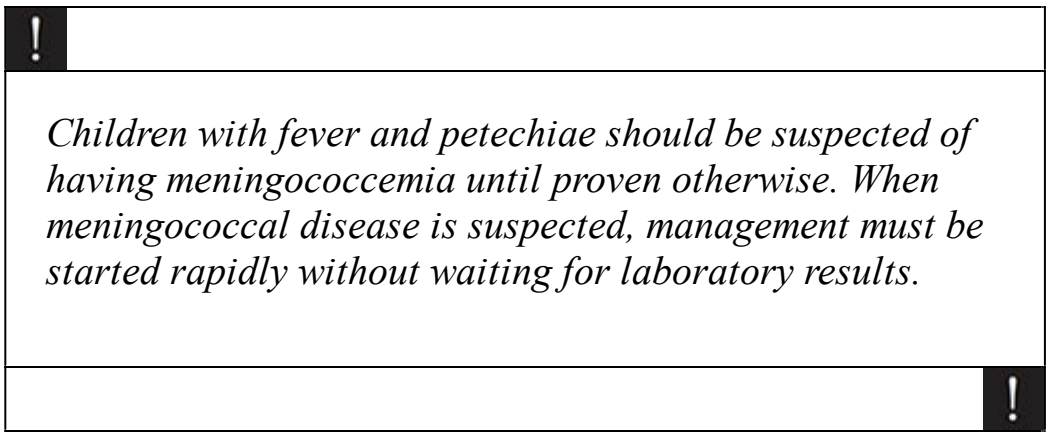
– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Neisseria meningitidis is a Gram-negative diplococcus that causes meningococcemia and meningitis, as well as other localized infections (such as pneumonia in adults) and rarely pericarditis, arthritis, peritonitis, and occult bacteremia. The main serogroups that cause the disease are A, B, C, Y, and W135. The two age groups at highest risk are infants younger than 2 years and adolescents/young adults aged 16 through 21 years. Crowded living environments with a high number of adolescents and young adults increases the rate of disease.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Children with fever and petechiae should be suspected of having meningococcemia until proven otherwise. When meningococcal disease is suspected, management must be started rapidly without waiting for laboratory results.
!

Meningococcemia is a severe illness that progresses to septic shock within 12 to 24 hours in most cases. Initial symptoms, which can be nonspecific and mimic a common viral illness, include fever, headache, vomiting, abdominal pain, and myalgia. The classic rash of petechiae, purpura, and ecchymosis can appear later in the disease, but the rash can take any form (eg, vesicular, blanching macules) or may be absent. Other signs of shock can occur, such as tachycardia, poor peripheral perfusion, tachypnea, oliguria, confusion, decreased consciousness, and evidence of multiorgan failure. Poor prognostic signs include coma, hypothermia, hypotension, leukopenia, and thrombocytopenia. Patients may also have meningitis. *N meningitidis* can be isolated from the blood and CSF, so appropriate cultures should be obtained in any patient presenting with signs of fever, petechial rash, and systemic illness. Samples for Gram stain and culture can also be taken from petechial skin lesions, sputum, synovial fluid, and other body fluids.

Meningococcal capsular antigen for capsular types A, C, Y, and W135 sampled from the CSF, blood, and urine is useful, especially in those who have received antibiotics before the samples were obtained; the yield for type B, the predominant subtype in the United States, Europe, and Australia-Pacific, is low. PCR for bacterial DNA detection is useful in those who have received antibiotics and has replaced capsular antigen detection in most centers.

Meningococcemia requires early, aggressive fluid resuscitation and hemodynamic and respiratory support as in other causes of septic shock. If patients are hypotensive despite adequate fluid resuscitation and inotropic and vasopressor support, administration of corticosteroids can be considered because of the concern for adrenal insufficiency. A third-generation cephalosporin (ie, cefotaxime or ceftriaxone) is an appropriate initial antibiotic choice, but therapy may be narrowed to penicillin G if the organism is susceptible. Antibiotic therapy should not be delayed if cultures cannot be obtained in a timely manner. Surgical debridement of ischemic tissue is occasionally required.

Chemoprophylaxis should be given to household contacts, childcare or nursery school contacts who had contact in the 7 days before onset of illness, or those exposed directly to the patient's oral

secretions through kissing, sharing utensils, or during close unprotected physical examination. Antibiotics used for chemoprophylaxis are rifampin, ciprofloxacin, or ceftriaxone (**Table 7-10**).

Table 7-10	CHEMOPROPHYLAXIS FOR HIGH-RISK CONTACTS OF INVASIVE MENINGOCOCCAL DISEASE	
Age	**Dose**	**Duration**
Rifampin[a]		
<1 mo	5 mg/kg, orally, every 12 h	2 days
≥1 mo	10 mg/kg (maximum 600 mg), orally, every 12 h	2 days
Adults	600 mg, orally, every 12 h	2 days
Ceftriaxone		
<15 y	125 mg, intramuscularly	Single dose
≥15 y	250 mg, intramuscularly	Single dose
Ciprofloxacin[a]		
≥1 mo	20 mg/kg (maximum 500 mg), orally	Single dose
Adults	500 mg, orally	Single dose
Azithromycin	10 mg/kg (maximum 500 mg)	Single dose

[a]Not recommended for use in pregnant women.
Classified using Bilukha OO, Rosenstein N. Prevention and control of meningococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR*. 2005;54:1-21. Centers for Disease Control and Prevention Web site. http://www.cdc.gov/mmwr/indrr_2005.html. Accessed April 17, 2013.

D. Rocky Mountain Spotted Fever

Case Study

A 9-year-old girl presents in September with a 3-day history of temperature to 39°C (102.2°F), severe headache, and a rash. She is not sexually active, and her last menstrual period was 2 weeks ago. She denies dysuria. Her pulse rate is 103 beats/min and blood pressure is 125/75 mm Hg. On exam, a maculopapular rash is noted on her extremities, trunk, and palms and soles, some of which is non-blanching. She also has conjunctival injection and tender muscles.

Detection

– What physiologic parameters concern you about this patient?

– What are the possible etiologies for this patient's condition?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Rocky Mountain spotted fever (RMSF) is an infection with the obligate intracellular coccobacillus, *Rickettsia rickettsii*, transmitted by the American dog tick. Most cases occur between April and September in the United States. This bacterium infects endothelial cells of the small vessels of all major tissues and results in a systemic vasculitis. The classic clinical tetrad of RMSF is fever, severe headache, muscle tenderness, and rash. The initial rash is a blanching macular-papular rash on the ankles and wrist that spreads centripetally to involve the trunk and spreads to the palms and soles. The rash evolves into a non-blanching petechial rash. Other clinical features are nausea, vomiting, abdominal pain, conjunctivitis, stupor, edema, meningismus, and coma. Laboratory features suggestive of RMSF include hyponatremia, hypoalbuminemia, anemia, and thrombocytopenia. The WBC count can be normal or elevated. If CSF is obtained, an elevated WBC count with monocyte predominance may be seen. The diagnosis is confirmed with rickettsial serological tests, but empiric antimicrobial treatment of a suspected case should not await these results because they are typically not available rapidly and the initial serology may be negative.

RMSF can be a life-threatening infection, and treatment should be initiated based on clinical suspicion. Early treatment yields very good outcomes. The drug of choice is doxycycline (or tetracycline); alternatively, chloramphenicol can also be used. Although staining of teeth by tetracycline is a concern, this is a dose- and duration-related complication, less likely with doxycycline than with tetracycline, and therefore unlikely to occur when the drug is administered for a short course. In light of the potential severity of this infection, administration of doxycycline should not be delayed.

E. Necrotizing Fasciitis

Case Study

A 15-year-old girl presents to the emergency department with marked pain in the left ankle, erythema, and swelling. She has a temperature of 39.4°C (102°F), is tachypneic, has an elevated heart rate, and the Spo_2 is 88% on room air. You are concerned that an apparent localized infection is presenting with systemic symptoms.

Detection

– What physiologic parameters concern you about this patient?

– What diagnosis are you most likely considering?

– What infectious etiologies are you concerned about with these findings?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Soft tissue infections consist of a spectrum of diseases, ranging from superficial (and more benign) infections (erysipelas, impetigo, or cellulitis) to life-threatening deep infections (necrotizing fasciitis and myonecrosis). Although necrotizing fasciitis is an uncommon disease, early recognition and treatment is critical for patient survival. GABHS is the most common individual bacterial species, but the microbe responsible for gas gangrene, *Clostridium perfringens*, is well described. Community-acquired methicillin-resistant *S aureus* is also increasingly common, but most infections are polymicrobial. There is no difference in outcomes between single-organism and polymicrobial infection. The necrosis of skin, underlying fat, and fascial layers leads to the clinical presentation of erythema, warmth, swelling, fever, and exquisite tenderness out of proportion to the findings of the rest of the physical exam. The overlying skin may develop crepitation as well as serous or hemorrhagic bullae. Patients may present in septic shock or multiple organ failure. Untreated,

necrotizing fasciitis is invariably fatal; therefore, a high index of suspicion must be maintained and treatment rapidly instituted. A diagnosis relies primarily on the clinical exam, but may be supported by magnetic resonance imaging (MRI) and laboratory values, such as increased values for C-reactive protein, WBC count, serum creatinine, and serum glucose, and decreased values for hemoglobin or serum sodium. A biopsy can confirm the diagnosis.

Early aggressive treatment, including early surgical consultation for surgical debridement, is necessary for patient survival. Initial antibiotic therapy should include broad-spectrum coverage of aerobic Gram-positive and Gram-negative organisms and anaerobes, such as is achieved with a carbapenem. For GABHS, high-dose parenteral clindamycin is optimal, with the addition of high-dose penicillin recommended by some, particularly in the presence of gas-forming organisms. If methicillin-resistant *S aureus* is suspected, then vancomycin or daptomycin should be used.

Additional therapies, such as high-dose IVIG, has proven beneficial in many cases. Hyperbaric oxygen may be considered as it may speed healing and limits tissue loss.

F. Bacterial Meningitis

Case Study

A 7-month-old boy is admitted to the pediatric ward with a 3-day history of an upper respiratory infection and a 1-day history of high fever, vomiting, and inconsolable irritability. He has not been feeding well and has had decreased urine output over the previous day. His heart rate is 148 beats/min, and his respiratory rate is 24 breaths/min.

Detection

– What physiologic parameters concern you about this patient?

– What diagnoses should you consider?

Intervention

– What additional tests are needed to confirm the diagnosis?

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

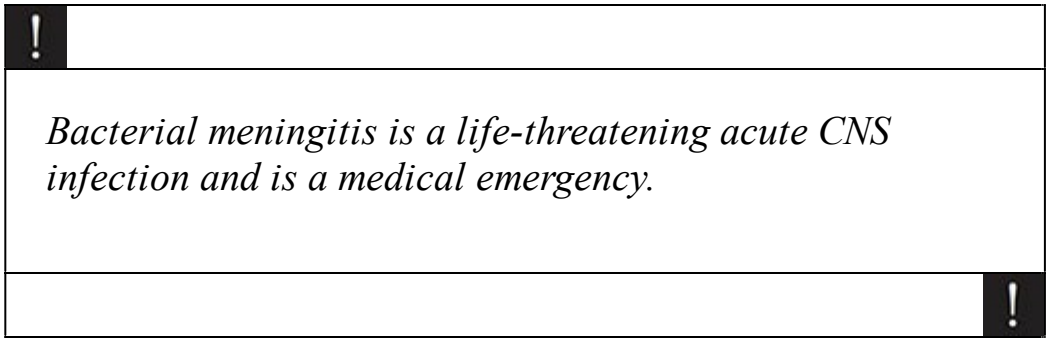
– How will the treatment strategy be implemented?

– Who is to do what and when?

Fever and vomiting without diarrhea can occur early in the course of a common infection, like acute gastroenteritis, but may be a sign of an intracranial infection, such as meningitis. Immediate identification of acute infections of the central nervous system (CNS) is very important due to the life-threatening or life-altering nature of untreated infections, although this task may prove difficult due to the varied presentations. Bacterial or viral meningitis, viral meningoencephalitis, or viral encephalitis account for most CNS infections in children. Typical symptoms of CNS infection include:

- Acute onset of fever
- Altered consciousness
- Headache
- Stiff neck
- Photophobia
- Nausea and vomiting
- Anorexia
- Seizures
- Bulging fontanelle

Often a child will have an antecedent upper respiratory tract infection. Children younger than 18 months with meningitis may not show signs of meningeal irritation (ie, neck stiffness, Kernig sign, Brudzinski sign), making it important to keep a high index of suspicion for meningitis or another CNS infection.



Bacterial meningitis is the most common life-threatening acute CNS infection in children and is a medical emergency. Common pathogens based on patient age are summarized in **Table 7-11**. The diagnosis of bacterial meningitis is made on the basis of clinical presentation and confirmed by the culture of pathogenic organisms in the CSF obtained by lumbar puncture. Circumstances in which a lumbar puncture may place the patient at risk and may be contraindicated are: cardiorespiratory instability, bleeding disorders, signs of raised intracranial pressure (irregular pupils, abnormal breathing pattern, hypertension, bradycardia, decerebrate or decorticate posturing), evidence of a soft tissue infection over the lumbar puncture site, recent (<30 minutes) or prolonged (>30 minutes) seizure, Glasgow Coma Scale score <13, the presence of papilledema, localized neurologic signs, and intracranial mass or hydrocephalus. A normal computed tomography (CT) scan does not rule out the possibility of cerebral herniation; CT of the head is not a prerequisite for performing a lumbar puncture. Also, the presence of an open anterior fontanelle does not protect infants from cerebral herniation. If a lumbar puncture is deferred, antibiotic therapy appropriate for bacterial meningitis should be administered immediately. Results of a CSF analysis consistent with bacterial meningitis, compared to other types of CNS infections, are described in **Table 7-12**. Blood cultures should also be obtained, as they are often positive for the offending organism.

Table 7-11 COMMON PATHOGENS CAUSING BACTERIAL MENINGITIS

Neonates	Group B streptococci *Escherichia coli* Other Gram-negative bacilli *Listeria monocytogenes*
Older infants and children	*Streptococcus pneumoniae* *Neisseria meningitidis* *Haemophilus influenzae* type b (in those not immunized)

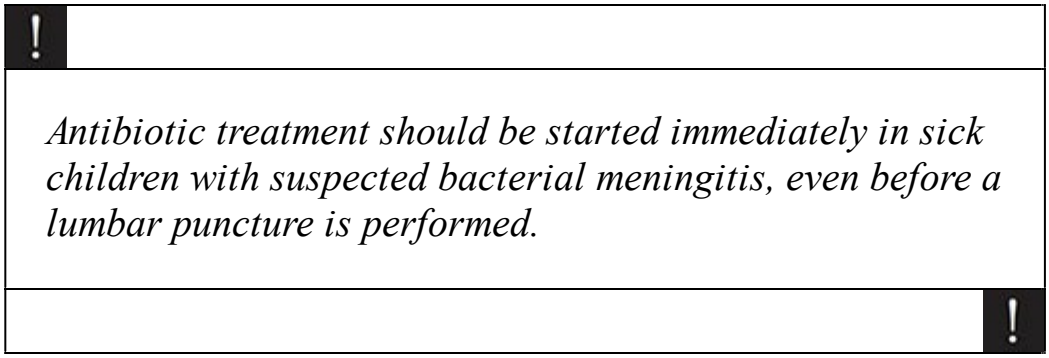
Table 7-12 CEREBROSPINAL FLUID FINDINGS IN MENINGITIS AND ENCEPHALITIS

Condition	Opening Pressure	Leukocytes (mm^3)	Glucose	Protein (mg/dL)
Bacterial meningitis	Elevated	100-10,000 (usually >1000); usually PMNs	Low (less than two-thirds normal serum glucose)	>40 (usually >200)
Viral meningitis	Normal to elevated	10-1000 (usually 100-500); usually lymphocytes	Usually normal	50-100
Tuberculous meningitis	Usually elevated	5-500; usually mononuclear cells	Normal to low	>500
Fungal meningitis	Usually elevated	5-500	Normal to low	100-500
Viral encephalitis	Normal to elevated	Normal to mild pleocytosis; either PMNs or mononuclear cells	Normal to mildly low	Normal to 100

PMN, polymorphonuclear leukocytes

Prompt administration of appropriate antibiotics in appropriate doses is the basis of curative therapy.

Empiric antibiotic choices are based on the patient's age and risk factors. In neonates, ampicillin combined with gentamicin or cefotaxime is the appropriate empiric therapy. In older infants and children, a third-generation cephalosporin (cefotaxime or ceftriaxone) combined with vancomycin should be started until bacterial identification and susceptibility have been established.


!
Antibiotic treatment should be started immediately in sick children with suspected bacterial meningitis, even before a lumbar puncture is performed.
!

Although controversial, the administration of corticosteroids (dexamethasone) early in the course of bacterial meningitis should be considered in children older than 8 weeks; it has been shown to decrease the incidence of hearing loss in patients with *Haemophilus influenzae* and *Streptococcus pneumoniae* meningitis. If prescribed, a dose of 0.15 mg/kg, given every 6 hours, should be administered for a total of 2 to 4 days, with the first dose given before or at the time of the first antibiotic dose. Chemoprophylaxis in cases of *H influenzae* type b (HIB) should be given to all household contacts when there is a susceptible contact in the home (eg, child <4 years of age who is incompletely immunized against this organism, child <12 months of age, or child who is immunocompromised). Chemoprophylaxis should also be given to day care center contacts in the presence of at least 2 cases of invasive disease within 2 months. Rifampin in a dose of 20 mg/kg once daily for 4 days (maximum dose, 600 mg) or 10 mg/kg/day in infants younger than 2 months is recommended. Pregnant women should receive ceftriaxone, 125 to 250 mg intramuscularly, as a single dose. Chemoprophylaxis for contacts of persons with meningococcal meningitis is the same as for meningococcemia.

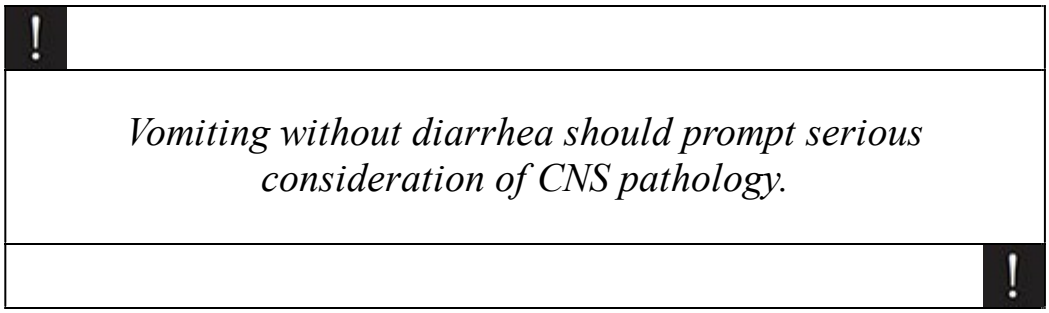
G. Aseptic and Viral Meningitis

Viruses are the causative agent in the majority of cases of "aseptic meningitis." Other etiologies are: *Mycoplasma pneumoniae*, Lyme disease, *Mycobacterium tuberculosis*, and RMSF. Parasitic causes (ie, *Angiostrongylus, Gnathostoma, Schistosoma, Toxocara, Echinococcus*) should also be considered in relevant geographic areas and returned travelers as they have a very different prognosis and are generally treatable. Rare noninfectious cases include malignancy, autoimmune disease, drug reaction, collagen vascular disease, or sarcoidosis (among other causes). Enteroviruses are the most common viral cause (especially echovirus and Coxsackie virus). Generally, the CSF cell count, differential, glucose concentration, protein concentration, and Gram stain results help differentiate between viral and bacterial causes, as the clinical presentations overlap. Although lymphocytes are the predominant cell type found in the CSF of patients with viral meningitis, a neutrophil predominance may be seen early in the course. Treatment is largely supportive, placing focus on airway, breathing, and circulation, as well as symptomatic therapy, such as control of pain, fever, seizures, and ensuring adequate hydration.

H. Encephalitis

Acute encephalitis is characterized by fever and changes in mental status. It is frequently caused by an enterovirus, herpes simplex virus (HSV), or another virus (ie, Epstein-Barr, cytomegalovirus, varicella-zoster virus, or an arbovirus). *M pneumoniae* and other etiologic agents may also be implicated. When children have recently been exposed to bodies of natural fresh water (ie, ponds, lakes), consideration of amebic encephalitis (caused by *Naegleria fowleri* or *Balamuthia mandrillaris*) should be entertained. In many cases, the etiology remains unknown despite investigations.

Results of CSF studies usually show a mild pleocytosis, but findings also may be normal. PCR of the CSF should be used to detect enterovirus and HSV infections. CNS imaging with MRI is more sensitive than CT and may detect areas of inflammation or help rule out noninfectious causes. For instance, during HSV encephalitis, the MRI may show alterations in the medial temporal lobe and/or frontal lobes, and the electroencephalogram may reveal typical periodic lateralized epileptiform discharges. In patients with suspected HSV encephalitis, urgent treatment with IV acyclovir should be initiated until this etiology can be ruled out. Data are insufficient to determine if the use of antibiotics in *M pneumoniae* encephalitis improves the clinical course or outcome.



Case Study

A 16-year-old girl presents to the emergency department with a 1-day history of a temperature to 40°C (104°F), abdominal and back pain, and an episode of vomiting. She is not sexually active and denies dysuria or discharge. On physical examination you note diffuse abdominal tenderness more pronounced on her left side. Her heart rate is 124 beats/min, and her respiratory rate is 22 breaths/min.

Detection

– What physiologic parameters concern you about this patient?

– What are the most likely, and most serious, diagnoses to consider?

Intervention

– What are the most immediate treatment strategies?

– What further investigation is needed to identify the etiology?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

The differential diagnosis of acute abdominal pain is extensive and involves many diagnoses that are not emergent. Both infectious and noninfectious illnesses within the abdominal compartment may need immediate attention due to their serious natures. The following noninfectious disorders can be acutely life-threatening and, among others, should be considered:

- Malrotation with volvulus
- Acute small or large bowel obstruction
- Intestinal perforation
- Acute kidney injury due to obstructive uropathy
- Viscus injury due to trauma
- Peptic ulcerative disease
- Acute pancreatitis
- Ectopic pregnancy
- Ovarian/testicular torsion

I. Urinary Tract Infection

Acute infections of the urinary tract can involve the bladder (cystitis) or the kidneys (pyelonephritis), and the latter may lead to bacteremia and sepsis (urosepsis). In children experiencing their first urinary tract infection (UTI), the most frequent pathogen is *Escherichia coli*. Other causative organisms are *Klebsiella, Proteus, Enterococcus faecalis*, and *Staphylococcus saprophyticus*. In

children who have a urinary tract obstruction, urinary catheter in place, or exposure to healthcare settings, *Pseudomonas aeruginosa* and extended spectrum β-lactamase-producing organisms (ie, *Klebsiella pneumoniae*, *E coli*) are possible pathogens.

The diagnosis of UTI is usually established with urinalysis and urine culture. A urinalysis that shows pyuria and a Gram stain that reveals bacteria can provide rapid evidence of the presence of a UTI. The "gold standard" diagnostic technique for obtaining urine is a suprapubic aspiration; when this technique is used, any bacterial growth is indicative of infection. However, urine for a culture is most frequently obtained using the midstream, clean-catch technique, intermittent catheterization, or a Foley catheter already in place.

It can be difficult to differentiate upper from lower UTI. Fever, leukocytosis, and flank tenderness may be present in patients with acute pyelonephritis. Urgency, frequency, and dysuria are classic symptoms of cystitis.

Children with a toxic appearance should always be treated with parenteral antibiotics. Cefotaxime or ceftriaxone is a good empiric choice in children and adolescents, while ampicillin and gentamicin are often prescribed for neonates. Extended spectrum β-lactamase- and metallo-β-lactamase-producing Gram-negative organisms are important causes of UTIs in some Asian countries and may require treatment with a carbapenem, such as meropenem. Neonates with UTIs should receive parenteral treatment because of the high prevalence of concomitant bacteremia.

J. Peritonitis

Depending on the source, peritoneal infections can be classified as primary or secondary peritonitis. Peritonitis is considered primary when the peritoneal cavity is infected via hematogenous or lymphatic dissemination and the cause is a single organism. The bacterial organisms that most frequently cause primary peritonitis are *Streptococcus pneumoniae*, *S pyogenes* (GABHS), and *E coli* or other enteric bacteria. The most frequent predisposing factor for primary peritonitis is the presence of ascites caused by nephrotic syndrome, but it can be due to cirrhosis and portal hypertension.

Secondary peritonitis results from the rupture or extension of an intra-abdominal viscus or abscess; therefore, it is usually polymicrobial. The most frequent pathogens are enteric Gram-negative bacteria, such as *E coli*, *Klebsiella*, or *Enterobacter*, plus anaerobes such as *Bacteroides fragilis*, and, at times, *Enterococcus*.

A patient suspected of having peritonitis should be managed with the consultation of a pediatric surgeon. Diagnostic techniques, such as ultrasound or CT scan, can help localize the source and extent of the infection. Empiric antibiotic treatment for primary peritonitis is usually cefotaxime or ceftriaxone in combination with an aminoglycoside. Antibiotic treatment for secondary peritonitis should be aimed at treating anaerobic and Gram-negative organisms. Agents used frequently are extended spectrum β-lactams like piperacillin/tazobactam, carbapenems like meropenem, ampicillin/sulbactam or ampicillin in combination with gentamicin or ceftriaxone, and metronidazole.

K. Acute Hepatic Failure

Acute or fulminant hepatic failure refers to the metabolic and systemic consequences of hepatic cellular dysfunction and necrosis, specifically including the hepatic encephalopathy and coagulopathy that develop within 8 weeks of onset of liver disease in a patient without preexisting liver disease.

Approximately 6% of cases of acute hepatic failure in the United States are due to primary viral infections, the second most common identifiable source of liver failure after acetaminophen toxicity. Responsible viruses are adenovirus, cytomegalovirus, Epstein-Barr virus, enterovirus, hepatitis A and hepatitis C viruses, and HSV. Worldwide, other organisms known to cause diffuse hepatic dysfunction are hepatitis B and hepatitis D viruses, echovirus, and leptospira.

Most patients with fulminant hepatic failure present with hepatic dysfunction, hypoglycemia, coagulopathy, and encephalopathy. Jaundice may be a late finding. Most patients have no history of other chronic medical problems or known exposure to hepatitis.

Management of fulminant hepatic failure is primarily supportive as no specific therapy exists other than liver transplant. Hepatic support, treatment of acquired infections, and prevention and treatment of complications are indicated while the patient awaits recovery of liver function. Extracorporeal hepatic support is often used as a bridge to liver transplant. Rapid referral to a transplant center, if an available option, is crucial, as patients may deteriorate rapidly, making them unsuitable for stable transfer. Broad-spectrum antibiotics are only indicated if sepsis or another bacterial infection is suspected.

L. Myocarditis and Pericarditis

Case Study

A 7-year-old boy presents to the emergency department with a 1-day history of increased work of breathing and worsening cough. One week earlier, he had been diagnosed with an upper respiratory infection consisting of a temperature to 38.4°C (101.2°F), cough, and nasal discharge. On presentation, his respiratory rate is 42 breaths/min, heart rate is 184 beats/min, and Spo_2 is 88% on room air. You hear crackles bilaterally on chest auscultation and feel a liver edge palpable 2 cm below the costal margin.

Detection

– What physiologic parameters concern you about this patient?

– What are the most likely, and most serious, diagnoses to consider?

Intervention

– What are the most immediate treatment strategies?

– What further investigation is needed to identify the etiology?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

Respiratory distress or failure is not always due to a primary pathology within the lungs. Primary myocardial failure (ie, cardiomyopathy) or inflammatory heart disease often presents with respiratory symptoms. Inflammatory heart disease encompasses a wide array of clinical entities, many of which are a result of acute infections of the pericardium and/or myocardium. Pericarditis may be caused by viral, bacterial, mycobacterial, or fungal infections. Viruses are the main cause of myocarditis. **Table 7-13** lists many of the infectious etiologies of pericarditis and myocarditis. Rheumatic fever, an immune-mediated illness that follows group A β-hemolytic streptococcal pharyngitis, may present with fever and carditis.

Table 7-13 INFECTIOUS CAUSES OF MYOCARDITIS AND/OR PERICARDITIS[a]

Viruses	Bacteria	Parasites	Fungi
• **Adenovirus** • **Coxsackie virus A, B** • **Echovirus** • **Parvovirus B19** • **Epstein-Barr virus** • Hepatitis B virus • Herpes simplex virus • **Human immunodeficiency virus** • Influenza virus A, B • Mumps virus • Poliovirus	• ***Borrelia burgdorferi*** • *Chlamydia psittaci* • *Corynebacterium diphtheriae* • *Leptospira* • Meningococcus • ***Mycoplasma pneumoniae*** • ***Staphylococcus aureus*** • *Streptococcus* sp • *Treponema pallidum* • *Actinomyces* • *Haemophilus influenzae* type b • *Mycobacterium tuberculosis*	• Echinococcosis • *Plasmodium falciparum* • Schistosomes • Toxoplasmosis • *Trichinella spiralis* • *Trypanosoma cruzi*	• *Candida* • *Coccidioides* • *Histoplasma* • *Aspergillus* • *Cryptococcus* • *Blastomyces*

[a]Some common etiologies of myocarditis or pericarditis are indicated in bold.

Patients with pericarditis may present with chest pain and fever. The pain is often substernal and may radiate to the middle of the back, increase with inspiration, and be relieved by leaning forward. If the

effusion that develops within the pericardial space becomes sufficiently large, patients may also have dyspnea, shortness of breath, shock, or respiratory failure. A friction rub may be heard on auscultation, but may be absent in the presence of a large effusion. A chest radiograph revealing an enlarged cardiac silhouette, an electrocardiogram showing ST- and T-wave changes or diminished QRS amplitudes supports the diagnosis, and an echocardiogram can confirm the diagnosis. Laboratory findings are nonspecific.

Myocarditis occurs when the myocardium is directly affected by an invading microorganism, often leading to inflammatory changes and myocyte necrosis. The spectrum of infectious etiologies is similar to that of pericarditis, but is most commonly viral in etiology. The range of presentations varies from an asymptomatic patient to a one with obvious signs of congestive heart failure. Many children will have a febrile illness preceding the episode of myocarditis. They may present with chest pain or syncope, but commonly the multisystem effects and symptoms of the underlying viral illness overshadow the symptoms related to the cardiovascular system. Infants may present with irritability and poor feeding. Dysrhythmias such as ventricular ectopy, atrioventricular block, ST- and T-wave abnormalities, or tachycardia out of proportion to the fever at presentation may suggest the presence of myocarditis. Sudden death is possible in a patient with severe acute myocarditis. The majority of laboratory and electrocardiographic findings are nonspecific. Elevation of troponin I is the most sensitive diagnostic marker other than myocardial biopsy results. Laboratory evidence of poor oxygen delivery due to poor cardiac output (ie, serum lactate) or myocardial stretch due to volume overload (ie, B-type natriuretic peptide) is often sought. The diagnosis is often difficult to establish and is generally made on circumstantial evidence, such as a recent viral infection and the sudden onset of cardiac dysfunction. Isolation of the infectious etiology should be pursued, but it is often elusive; use of PCR may increase the likelihood of success.

Myocarditis and pericarditis therapies include treating the infecting pathogen (for which a treatment is available), as well as providing medical support of myocardial function with a combination of afterload reduction (ie, milrinone), inotropy (ie, dopamine or epinephrine [adrenaline]), and diuretics (ie, furosemide). Severe myocardial dysfunction may also necessitate mechanical ventilation, either noninvasively with bilevel positive airway pressure or invasively with intubation. Rapid deterioration of myocardial function can occur in acute fulminant myocarditis, warranting serial echocardiography and, if evident, transfer to a pediatric cardiac transplantation center if possible. If a large pericardial effusion is present and impinging on cardiac function or if purulent pericarditis is present, drainage is indicated. IVIG is indicated in the treatment of viral myocarditis.

M. Neonatal Herpes Simplex Virus Infections

Case Study

A 4-day-old, full-term healthy neonate is admitted to the hospital with a history of a temperature to 40°C (104°F). Her physical examination appears normal, including the skin. The infant was born via normal spontaneous vaginal delivery after 18 hours of ruptured membranes. The maternal history

revealed no known risk factors. A full sepsis workup is done, and the infant is started on ampicillin and cefotaxime. Despite antibiotics, she develops respiratory distress and hypotension.

Detection

– What physiologic parameters concern you about this patient?

– What are the most likely, and most serious, diagnoses to consider?

Intervention

– What are the most immediate treatment strategies?

– What further investigation is needed to identify the etiology?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

In addition to bacterial infection, two viral etiologies should be entertained in a newborn presenting with multisystem involvement with or without hypotension: HSV infection and enterovirus sepsis.

Neonatal HSV is most commonly acquired in the peripartum period. Approximately 50% of cases are due to HSV-1 and the remaining to HSV-2. Of the affected infants, 50% to 70% are born to women who have asymptomatic infection at delivery. The highest risk of infection (~50%) is in infants born to women with primary infection during pregnancy.

Neonatal HSV can present in 3 forms:

1. Disseminated disease accounts for approximately 25% of these infections. It usually presents in the first 2 weeks of life with multisystem involvement, including the lungs, liver, adrenal glands, skin, eye, and/or brain. The mortality rate is high, and death is usually due to severe coagulopathy, liver failure, and/or respiratory failure.
2. CNS disease without skin lesions presents later than 2 weeks and manifests as fever, irritability, poor feeding, and seizures, which can be either focal or generalized. This form accounts for

approximately 25% of neonatal HSV cases.

3. Disease limited to the skin, eye, and/or mouth occurs in 50% of neonatal HSV infections. These neonates should have a CSF evaluation as one-third have CNS involvement.

Typical vesicular skin lesions can occur at any time in the course of illness, but 10% to 20% of infants with either disseminated infection or CNS involvement do not develop lesions. This makes the initial diagnosis difficult as the signs and symptoms of sepsis caused by other etiologies are similar and nonspecific. A high index of suspicion should be maintained in a neonate with either sepsis or seizures. To make the correct diagnosis, swabs for viral culture or PCR using appropriate transport media should be collected from any suspicious lesions, as well as surface cultures from the mouth, nasopharynx, conjunctivae, and rectum. CSF evaluation should be performed routinely and include PCR for HSV. Blood also should be tested by PCR.

Treatment consists of high-dose acyclovir at 60 mg/kg/d divided every 8 hours. The duration of disseminated and CNS disease is at least 21 days, and 14 days for skin, eye, and/or mouth disease without CNS involvement.

N. Lemierre Disease (Necrobacillosis)

Case Study

You are caring for a 16-year-old previously healthy boy who developed a sore throat and malaise about 5 days before admission. After 2 days, he began having pain in the right side of his neck and had difficulty turning his head from side to side. He also reports chills with fever, pleuritic chest pain, and abdominal pain. He appears ill, is tachypneic, and febrile to 39°C (102.2°F). His pharynx appears normal, and he has a tender, ill-defined fullness in the right sternocleidomastoid muscle. His lung and heart exams are normal. Multiple bilateral nodular infiltrates are seen on chest radiograph.

Detection

– What physiologic parameters concern you about this patient?

– What are the most likely, and most serious, diagnoses to consider?

Intervention

– What are the most immediate treatment strategies?

– What further investigation is needed to identify the etiology?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

Originally described in the early 20th century and also referred as to as *postanginal sepsis* or *necrobacillosis*, Lemierre disease is thought to be caused by the spread of infection from the oropharynx into the parapharyngeal space, resulting in septic thrombophlebitis of the internal jugular vein, bacteremia, septic emboli and, at times, septicemia. This illness typically affects adolescents and young adults. Most patients present with fever and a sore throat, followed by neck pain that can be associated with neck swelling, trismus, and dysphagia. Septic emboli most often spread to the lungs, and these patients present with pneumonia with empyema. Chest radiographs can show bilateral nodular infiltrates, cavitary lesions, effusions, or pneumatoceles. Dissemination to other organs—such as the musculoskeletal system (causing septic arthritis and osteomyelitis) or the liver, spleen, and kidneys—is possible. Occasionally retrograde spread to the brain can cause cerebral venous sinus thrombosis, meningitis, or brain abscess.

The most likely causative organism is the anaerobe *Fusobacterium necrophorum* or another *Fusobacterium* sp, but other organisms have also been reported, such as anaerobic streptococci, *S aureus*, and *Bacteroides* sp. Both anaerobic and aerobic blood cultures should be obtained in adolescents older than 11 years with a febrile illness and a suspicion for Lemierre disease. Imaging of the neck also should be performed to detect thrombophlebitis of the internal jugular vein. Ultrasound is useful if positive but is not very sensitive; therefore, either a CT or MRI scan may be performed to establish the diagnosis.

Patients with Lemierre disease should receive a penicillin/β-lactamase inhibitor agent (such as piperacillin/tazobactam) or a combination of ceftriaxone and clindamycin to treat both *Fusobacterium* and streptococci that might be present. Metronidazole is active against all *Fusobacterium* sp and is often used with a β-lactam. The duration of antibiotic treatment is usually 4 to 6 weeks. Anticoagulation therapy has been used in both adults and children with jugular vein thrombosis and cavernous sinus thrombosis. This may decrease the risk of clot extension and shorten recovery time, although its use is controversial due to a lack of definitive data. Surgical intervention involving debridement or incision and drainage is occasionally necessary.

O. Malaria

Case Study

A 12-year-old boy presents to the emergency department with a fever of 7 days' duration; it developed shortly after his return from a 3-week trip to Ghana. He initially had flu-like symptoms, and he reports vomiting and diarrhea with progressive confusion for the last 3 days. He experienced an episode of generalized tonic-clonic seizure on the day of admission. His temperature is 39.3°C (102.7°F), pulse is 129 beats/min, blood pressure is 112/73 mm Hg, and respiratory rate is 32 breaths/min. Physical examination reveals hepatosplenomegaly, jaundice, slurring of speech, disorientation, and an inability to follow commands. His laboratory results include the following: WBC/ 10.7 mm^3 (65% neutrophils, 20% bands); hemoglobin, 8.1 g/dL; and platelets, 47,000/mm^3.

Detection

– What physiologic parameters concern you about this patient?

– What are the most likely, and most serious, diagnoses to consider?

Intervention

– What are the most immediate treatment strategies?

– What further investigation is needed to identify the etiology?

Reassessment

– Is the current treatment strategy effective?

– Are there any adjunctive therapies to consider?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best location to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

In any patient with a significant travel history, it is necessary to consider and recognize certain infections endemic to specific regions of the world, as morbidity and mortality rates may be high.

!

Malaria should be excluded in any traveler with fever, even if malaria chemoprophylaxis was administered, as it

is not 100% protective.

Malaria is transmitted by the bite of the female *Anopheles* mosquito. Of the 5 *Plasmodium* species that cause malaria in humans, *P falciparum* results in the most severe disease. Infection with *P falciparum* is potentially fatal and is responsible for 40% to 60% of the world's cases of malaria and for 95% of deaths from malaria. Complications include severe anemia, cerebral malaria, and pulmonary edema. *P vivax* and *P ovale* can be dormant in the liver, and illness can occur as long as several years after initial infection or can relapse. *P malariae* can lead to chronic, long-term illness. The newly described *P knowlesi* is largely confined to Indonesia and Malaysia; its presentation is similar to *P falciparum* malaria, and infection may be fatal. Knowledge of travel destinations and distribution of *Plasmodium* species is important in the selection of antimalarial drugs. Dual infection is possible and, in fact, common in some areas.

The initial presentation of malaria is nonspecific and can include fever, chills, headache, vomiting, diarrhea, myalgias, weakness, dizziness, and splenomegaly. The classic cyclic fever every second day (*P falciparum*, *P vivax*, and *P ovale*) and every third day (tertian fever) is infrequently observed in children. The physical exam may include hepatosplenomegaly, jaundice, diaphoresis, and pallor.

The diagnosis is based on demonstration of parasites in thick and thin blood smears. A single negative smear does not rule out malaria, and multiple smears should be done several hours apart if the index of suspicion is high. PCR for the malaria parasite is highly sensitive and specific, and can reveal both the presence of the parasites and the species involved. Other laboratory abnormalities are a low hemoglobin value, low platelet count, hyperbilirubinemia, and increased hepatic enzymes. Severe malaria is characterized by altered consciousness, seizures, anemia, oligoanuria/acute kidney injury, acidosis, and hypoglycemia and is a medical emergency. In determining the initial choice of antimicrobial therapy when the species is unknown, it is prudent to assume that the infection includes *P falciparum* and to treat accordingly. Choice of drug depends on several factors, including type of *Plasmodium* species, drug susceptibility of the infecting parasite, severity of illness, and availability of medications. Oral medications include artemether and artemisinin combination therapy, chloroquine, atovaquone-proguanil, mefloquine, and quinine sulfate plus doxycycline, tetracycline, or clindamycin. Chloroquine can be used in patients from areas where drug resistance has not been reported. Details of treatment are available at World Health Organization (http://www.who.int/malaria) and Centers for Disease Control and Prevention (http://www.cdc.gov/malaria/) websites.

Severe malaria in anyone other than women in the first trimester of pregnancy should be treated with the usual resuscitative measures in conjunction with IV artesunate, 2.4 mg/kg immediately, repeated at 12 and 24 hours, then once daily until oral therapy is available. Oral therapy is usually lumefantrine, amodiaquine, sulfadoxine-pyrimethamine, or mefloquine. Oral artemisinin combination therapies are available (eg, artemether-lumefantrine). Rectal administration is also an option in the comatose patient in whom IV and intraosseous access is not possible. Artemisinin compounds must not be given alone due to the high risk of inducing resistance. Women in the first trimester of pregnancy should receive IV quinine or quinidine, which requires admission to the ICU for cardiac monitoring. Exchange transfusion should be considered in the most severe cases (ie, *P falciparum* with >10%

parasitemia and end-organ damage) as a means of removing parasitized red blood cells.

P. Dengue Fever

Dengue fever is an acute febrile illness caused by one of four main dengue virus serotypes (DENV), transmitted by female *Aedes aegypti* and *Aedes albopictus* mosquitoes. The typical incubation period is 4 to 7 days. Patients can present with a mild febrile illness or the classic dengue fever (breakbone fever). Signs consist of fever and headache; severe muscle, joint, bone (back) pain; nausea and vomiting; rash; and mild bleeding. The rash can be macular, scarlatiniform, or petechial, sparing the palms and soles. In most cases, dengue is a self-limited illness, lasting 3 to 10 days. The most severe form of illness, dengue hemorrhagic fever/dengue shock syndrome, is a life-threatening condition. Warning symptoms of severe disease are recrudescence after initial improvement, severe abdominal pain, persistent vomiting, and development of hypothermia, hemorrhage, or change in mental status. Diagnosis of a dengue viral infection requires PCR or serial immunoglobulin M antibody titers, although the latter are notoriously difficult to interpret due to significant cross-reactivity with other flaviviruses.

The treatment of dengue fever is mainly supportive, with fluid resuscitation and hemodynamic support paralleling that of septic shock. Close monitoring of blood tests is necessary.

Key Points

Acute Infections

- Detection of potentially severe infections is based on a meticulous clinical examination and attention to vital signs.
- Adequate cultures should be obtained before starting antibiotic treatment, but initiation of antibiotics should not be unduly delayed.
- The choice of antibiotics depends on the patient's age, the type of infection suspected, risk factors (such as immunosuppression, presence of invasive medical devices, or recent hospitalization), and knowledge of local antimicrobial resistance patterns.
- Severe infections and localizing signs of infection can be difficult to detect in young children, infants, and neonates. Management is based on risk factors, toxic appearance criteria, and laboratory findings.
- Suspicion of sepsis requires rapid management with goal-directed therapy. Antibiotic treatment should be started within the first hour.
- *S aureus* and *S pyogenes* can produce toxin-mediated diseases, such as toxic shock syndrome.
- Meningococcemia should be considered in all children with fever and petechiae.

- Bacterial meningitis should always be treated emergently. CSF examination is often diagnostic, but initiation of antimicrobial therapy should not be delayed if there are contraindications for lumbar puncture.
- Acyclovir treatment for HSV encephalitis should be considered in patients with encephalitis, such as those with fever and alterations in consciousness.
- Nosocomial infections should be suspected in hospitalized children with fever. The most frequent bacteria involved include coagulase-negative staphylococci and Gram-negative enteric rods, which may exhibit resistance to multiple antimicrobials.
- Infected lines or medical devices should be removed.
- Infections endemic to certain geographic areas, such as dengue and malaria, may be associated with a high mortality rate if not treated adequately. Such infections should always be considered in patients who have recently traveled.

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FLUIDS, ELECTROLYTES, AND NEUROENDOCRINE METABOLIC DERANGEMENTS

Objectives

- Summarize basic concepts in the maintenance of intravascular fluid homeostasis.
- Discuss the common electrolyte disturbances, their recognition, and management.
- Describe common neuroendocrine emergencies, including those related to glucose metabolism, and discuss their management.

Case Study

A 9-month-old infant is brought to the emergency department with a 2-day history of vomiting, diarrhea, and fever. She is lethargic, irritable, confused, disoriented, and exhibits a sunken fontanelle. Laboratory results are as follows: Na^+, 125 mEq/L (125 mmol/L); K^+, 4.8 mEq/L (4.8 mmol/L); Cl^-, 89 mEq/L (89 mmol/L); glucose, 89 mg/dL (4.9 mmol/L); CO_2, 7 mEq/L (7 mmol/L); blood urea nitrogen, 50 mg/dL; creatinine, 1.2 mg/dL (106.08 μmol/L).

Detection

– What are the most important clinical signs?

– What are the most likely diagnoses?

Intervention

– After initial fluid boluses with isotonic saline, what is the plan for ongoing fluid management?

– If the sodium level drops to 117 mEq/L and the patient begins to seize, how will you respond?

Reassessment

– What is the anticipated response to the intervention?

– Is the current treatment strategy effective?

– What are the therapeutic endpoints to watch?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– How would you assign roles and prioritize tasks?

I. FLUIDS

Fluid, electrolyte, and endocrine-metabolic disturbances are commonly seen in hospitalized children. An individualized approach to the fluid management of these children is needed based on the underlying diagnosis and severity of illness.

A. Maintenance Fluids

Hypotonic and isotonic solutions are defined as follows:

- Isotonic solution – equal osmolarity compared with another solution
- Hypotonic solution – lower osmolarity compared with another solution
- Normal plasmatic osmolarity – between 280 and 300 mOsm/L

The fluid intake required to maintain a euvolemic state is largely dependent on the patient's initial hydration status and underlying disease state.

The term *maintenance fluids* loosely denotes the quantity of IV fluids required to maintain hydration in an euvolemic child who is not experiencing abnormal ongoing loss of fluids, as seen in gastrointestinal diseases, bleeding, and burns. Maintenance fluid calculations provide a rapid and convenient starting point to determine the amount of fluid required to maintain intravascular volume.

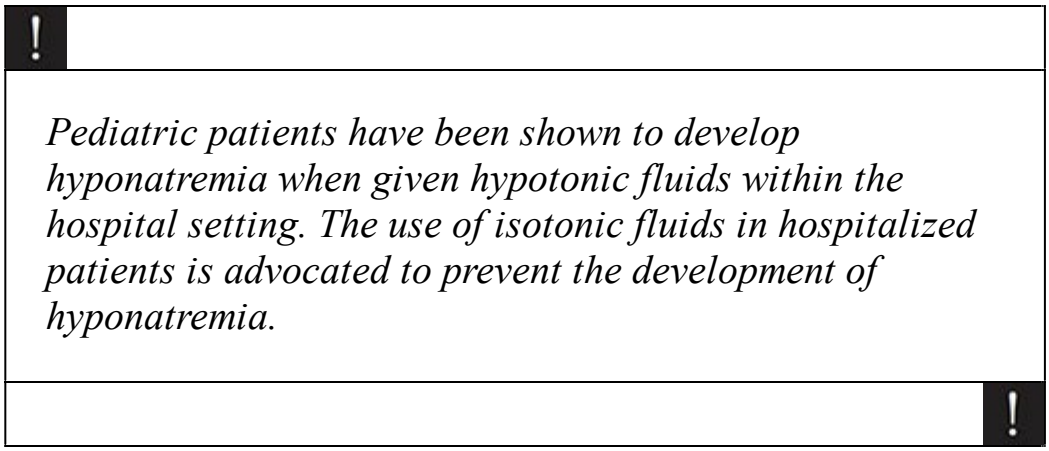
Hospitalized children are at risk for hyponatremia, and the use of hypotonic saline solutions is the main risk factor for this condition. Isotonic saline solutions have been shown to have a protective effect against hyponatremia and no significant deleterious effects, such as water overload, hypernatremia, or phlebitis. A 0.9% normal saline solution is the preferred maintenance solution for the ill child.

The Holliday-Segar method for calculating maintenance IV fluids is based on the caloric expenditure in fixed weight categories. Calculations are based on the patient's dry/baseline weight. Evidence indicates that the traditional Holliday-Segar recommendation for maintenance fluid therapy for sick and hospitalized children deserves to be reconsidered because of the associated adverse effects, as

well as the better results obtained with the use of isotonic solutions.

Other methods are available and utilize nomograms to determine fluid requirements based on body surface area and caloric expenditure.

IV fluids containing potassium should be withheld until the patient has satisfactory urine output to avoid a risk of inducing hyperkalemia. The general practice of adding 20 mEq/L KCl to the IV fluid solution and a "one-size-fits-all" approach to KCl concentrations in IV fluids should be avoided. The amount of KCl in the IV fluid should be individualized to each patient as urine and stool output, diuretics and other medications, as well as other parameters can affect potassium requirements.


!
Pediatric patients have been shown to develop hyponatremia when given hypotonic fluids within the hospital setting. The use of isotonic fluids in hospitalized patients is advocated to prevent the development of hyponatremia.
!

B. Resuscitation Fluids

Resuscitation fluids in infants and children remain exclusively isotonic. Initial recommendations for treating hypovolemia include 20 mL/kg boluses of normal saline. Reassessment and repeat boluses must be done quickly (up to 60 mL/kg in 30-60 minutes). Most resuscitation protocols suggest that initial fluid resuscitation should begin with crystalloid (normal saline or Ringer lactate). After 60 mL/kg of crystalloid has been administered, the use of colloid should be considered.

C. Fluid Resuscitation in Shock

Hypovolemia remains the most common cause of shock worldwide due to gastroenteritis. Rapid restoration of intravascular volume with fluid replacement remains the key to survival. Patients with inadequate or delayed fluid resuscitation are at risk of developing organ dysfunction or progressing to irreversible shock. Regardless of the type of shock, some degree of hypovolemia is usually present and IV fluid therapy should always be considered in the initial resuscitative measures. Each bolus should be administered within 5 to 10 minutes or as rapidly as permitted by vascular access. An exception to this is cardiogenic shock; these patients tend to be exquisitely sensitive to volume overload, and judicious administration of isotonic fluid, 5 to 10 mL/kg, is indicated, ideally with central venous pressure monitoring where feasible.

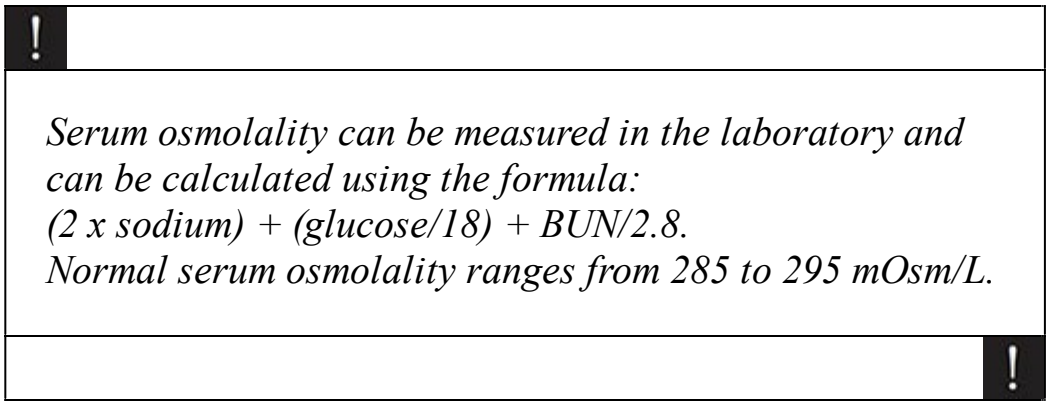
Rapid IV or intraosseous access should be established to provide a means of delivering the fluid and medications required during resuscitation.

Utilizing the DIRECT algorithm (**Chapter 1**), reassessment of vital signs and clinical status must be

conducted between boluses to evaluate for changes in clinical status and response to therapy.

II. ELECTROLYTE ABNORMALITIES

Electrolyte abnormalities are common in children. Occasionally these abnormalities are due to iatrogenic causes. Infants and children with severe electrolyte disturbances can present with poor feeding, irritability, dehydration, shock, altered mental status, and/or seizures. These disturbances may progress to coma or even death.


!
Serum osmolality can be measured in the laboratory and can be calculated using the formula:
(2 x sodium) + (glucose/18) + BUN/2.8.
Normal serum osmolality ranges from 285 to 295 mOsm/L.
!

A. Sodium

1. Hyponatremia

Hyponatremia is defined as a serum sodium level <135 mmol/L (<135 mEq/L). The sodium level can be decreased due to a variety of causes: increased water intake (dilute infant formula, polydipsia, infants drinking pool water), increased water retention (syndrome of inappropriate antidiuretic hormone [SIADH], renal failure), and increased sodium losses (gastroenteritis, diuretics, cerebral salt wasting, adrenal insufficiency).

Symptoms of hyponatremia include: irritability, poor feeding, nausea and vomiting, lethargy, seizures, and eventually coma and death. Hyponatremia may occur because of significant losses due to gastroenteritis, increased ostomy output, excessive sweating, cystic fibrosis, heatstroke, burns, or third spacing in pancreatitis or pleural effusions. Children treated with diuretics are frequently hyponatremic. Renal diseases such as renal tubular acidosis can cause loss of salt and water. In hospitalized children, excess secretion of antidiuretic hormone leads to the development of SIADH. Infants may present in the first few months of life with severe hyponatremia secondary to water intoxication from overly diluted formula or from swallowing large amounts of water in swimming or wading pools. Other causes include postobstructive uropathy and losses secondary to ventriculostomy drainage. Clinically, hyponatremia can be classified according to the patient's extracellular fluid volume status: hypovolemic, euvolemic, or hypervolemic (**Table 8-1**).

Table 8-1 Classification of Hyponatremia According to Extracellular Fluid Volume Status

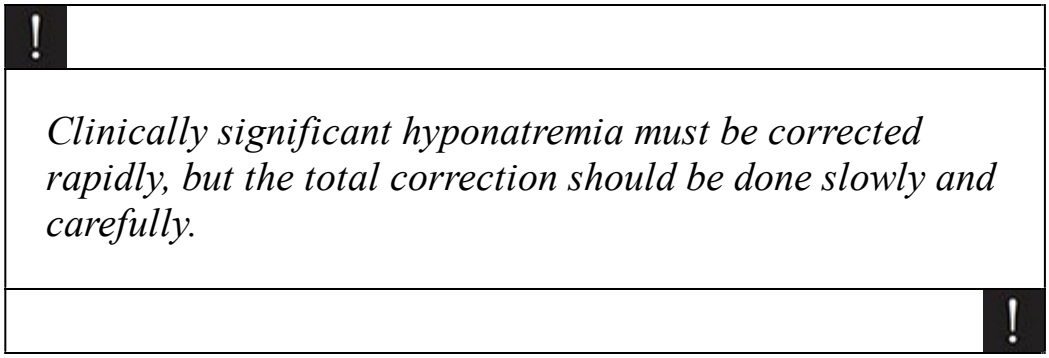
Euvolemia	Hypervolemia	Hypovolemia

SIADH	SIADH	Diarrhea
Adrenal insufficiency	Congestive heart failure	Vomiting
Central nervous system disease	Renal failure (acute/chronic)	Burns
Pulmonary disease	Nephrotic syndrome	Pancreatitis
	Cirrhosis	Cerebral salt wasting

SIADH, syndrome of inappropriate antidiuretic hormone

Urgent treatment of hyponatremia should be initiated in all patients who exhibit neurological changes or seizures and if the serum sodium level is <120 mEq/L. Most frequently, 3% NaCl (513 mmol/L) is used, if available. Ideally, 3% saline should be given through a central venous line; administration through a peripheral IV or an intraosseous needle is acceptable during an emergency while awaiting placement of central venous access. The goal is to raise the serum sodium level to between 120 and 125 mEq/L or until seizures stop. To raise the level, administer the calculated amount of hypertonic saline over 15 to 20 minutes to gain rapid control of the seizures. A 1.2 mL/kg aliquot of 3% NaCl will raise the level by 1 mEq/L. If hypertonic saline solution is unavailable, a bolus of normal saline (0.9% NS), 20 mL/kg, may be given.

Once the acute correction is completed or if the patient is not having neurological changes, correction of hyponatremia should proceed more slowly at approximately 12 mEq/L per day (0.5-1 mEq/L every hour). In adults, the syndrome of central pontine myelinolysis (osmotic demyelination) has been described in those who had a rapid increase in serum sodium values.



Clinically significant hyponatremia must be corrected rapidly, but the total correction should be done slowly and carefully.

The following formula is used to calculate the amount of sodium required to correct to a desired level (mEq/L):

$$0.6 \times (\text{weight in kg}) \times (\text{target } Na^+ - \text{measured } Na^+) =$$
$$\text{total mEq of } Na^+ \text{ required to raise sodium level to target}$$

Figure 8-1 illustrates how to use this formula in a clinical scenario. A good fluid for starting replacement is either normal saline or 5% dextrose in normal saline, with or without appropriate potassium at maintenance fluid rates and with frequent electrolyte checks.

Figure 8-1. Sample Calculation for Determining the Amount of Sodium Required

Case

A 1-year-old child (10 kg) presents with seizures and is found to have a sodium level of 114 mEq/L.

↓

First Step: Acute Correction

You decide to give 3% saline to correct to 120 mEq/L.

↓

Amount of NaCl = 0.6 x 10 kg x (120 -114) = 36 mEq of Na^+
3% NaCl = 0.5 mEq/L or approximately 28 mL of 3% NaCl solution

OR

(1.2 mL/kg) of 3% NaCl raises the serum sodium level approximately 1 mEq/L
1.2 mL x 10 kg x (120-114) = 72 mL of 3% NaCl solution

↓

Second Step: Slower Correction

You need to raise the serum sodium level an additional 12 mEq over the next 24 hours from the current 120 mEq/L.

0.6 x 10 kg x (132 desired Na^+ – 120 actual Na^+) = 72 mEq/L of additional sodium needed over the next 24 hours

a. Syndrome of Inappropriate Antidiuretic Hormone

By definition, SIADH is caused by an *inappropriate* release of antidiuretic hormone. When patients are hypertonic or significantly hypovolemic, antidiuretic hormone is released appropriately, with a resultant decrease in urine output to maximize the circulating intravascular volume. In patients with SIADH, the intravascular volume is normal or elevated and yet the urine output drops off. The diagnosis of SIADH requires three elements: low urine output, hyponatremia, and inappropriately concentrated urine (with elevated urinary sodium).

!
SIADH is the most common cause of hyponatremia in the hospitalized patient.
!

The causes of SIADH fall into four groups: central nervous system (CNS) abnormalities (eg, infection, trauma, surgery, tumors, shunts, hypoxic-ischemic injury) [Chapter 17]; pulmonary problems (eg, pneumonia, effusions, positive pressure ventilation, asthma, tumors); drugs (eg, carbamazepine, vinca alkaloids, narcotics, aspirin, Ecstasy, selective serotonin reuptake inhibitors); and tumors (eg, leukemia, lymphoma, neuroblastoma).

The treatment of SIADH is water restriction. Reducing water intake to 50% of maintenance fluid will often allow a free water diuresis and improve the serum sodium measurement. It is occasionally necessary to correct the sodium level with salt supplementation and diuretic administration.

b. Hyponatremia Due to Water Intoxication

Water intoxication occurs when powdered infant formula is diluted with an excessive amount of water, either inadvertently (not following directions properly) or purposely (to make the formula last longer), resulting in hyponatremia. This also is seen when formula or an electrolyte solution is being diluted when an infant has gastroenteritis and diarrhea.

Infant swimming is another potential source of hyponatremia. It follows after ingestion of large amounts of water when the infant is playing in a bathtub, wading pool, or splash tub. The infant presents with irritability and poor appetite. A targeted history and check of the serum sodium level reveal the cause.

2. Hyponatremia and Hypovolemic States

a. Nonrenal

Worldwide, gastroenteritis is the leading cause of death in children between 1 and 5 years of age. Typically the history is that of large and frequent watery stools. The patient may present in various stages of dehydration and possibly hypovolemic shock. Depending on the severity of the hypovolemia, the patient may require aggressive and timely fluid resuscitation with isotonic fluids.

Sepsis and peritonitis are examples of conditions where fluid can leak into the interstitial space (third spacing). This can occur fairly rapidly over a matter of hours, leading to hypovolemia and hyponatremia because water and electrolytes move into the interstitial spaces. Patients present with clinical evidence of shock and hypovolemia. Serum electrolyte measurements reveal hyponatremia, potentially hypokalemia and hypocalcemia.

Burns result in rapid and excessive loss of fluid due to the capillary leak phenomenon. Fluid losses are both external (across the surface area involved in the burn) and internal (into the interstitial spaces). Rapid and ongoing fluid resuscitation is required in the management of burns and thermal injuries.

b. Renal

Diuretics can lead to hyponatremia with both short- and long-term use. Loop diuretics, such as furosemide and bumetanide, and drugs that interfere with renal tubular reabsorption, such as metolazone, can lead to significant hyponatremia. Other renal causes of hyponatremia are renal tubular acidosis, interstitial nephritis, and obstructive uropathy.

c. Other Causes of Hyponatremia

The exact pathophysiology of cerebral salt wasting (CSW) is poorly understood. Hyponatremia occurs in the setting of normal to high urine output, and urinary sodium excretion is elevated. Patients tend to be hypovolemic and exhibit symptoms of dehydration and hyponatremia. The condition is seen in conjunction with intracranial injury, intracranial tumors, and neurosurgical procedures. It is important to distinguish CSW from SIADH, which also presents with hyponatremia but without the resultant hypovolemia seen in CSW. Treatment of SIADH includes fluid restriction, whereas fluid and

electrolyte replacement for the correction of hyponatremia is the therapeutic approach in CSW. Urinary sodium also tends to be higher in CSW (may be >100 mEq/L) than in SIADH.

Congestive heart failure and nephrotic syndrome are both examples of hypervolemic conditions associated with hyponatremia despite an increase in total body sodium content. The hyponatremia is dilutional in these cases, and sodium intake restrictions are an important part of management. Treatment is directed toward the underlying condition: diuretics and inotropes for the heart failure, steroids and diuretics in nephrotic syndrome, and dialysis in renal failure.

3. Hypernatremia

Hypernatremia is defined as a serum sodium >145 mEq/L. Groups at risk for elevated sodium are infants, toddlers, and critically ill patients. Hypernatremia occurs when too much salt is ingested or too much free water is lost. Children who develop hypernatremia may have received concentrated formula, been breast-fed without supplementation, or been treated with salt supplements or sodium bicarbonate. Free water loss can be due to diarrhea, diabetes insipidus (central or nephrogenic), renal tubular disorders, or postobstructive diuresis.

Signs and symptoms of hypernatremia include irritability, high-pitched cry, lethargy, seizures, fever, renal failure, and rhabdomyolysis. In infants, these symptoms mimic those of infections and sepsis.

It is essential that hypernatremia be corrected gradually with frequent monitoring of electrolytes. When the sodium level rises, the serum osmolality increases, and fluid moves from inside the cells to the serum in an attempt to achieve balance. This increases the osmolality inside the cell. Rapid administration of free water results in movement from low osmolality (ie, serum) to higher osmolality (ie, inside the cell), causing the cell to swell. This can result in cerebral edema.

Most recommendations propose lowering serum sodium values no more than 0.5 mEq/L/h or 12 mEq/L/day. The formula for calculating the amount of free water necessary to correct the serum sodium level is:

$$\text{Free water deficit} = (\text{weight in kg} \times 0.6) \times 1 - (\text{desired } Na^+/\text{actual } Na^+)(1000 \text{ mL/L})$$

- *1 L of 0.45% normal saline = 500 mL of free water*
- *1 L of 0.225% normal saline = 750 mL of free water*
- *1 L of dextrose 5% with 0.45% normal saline = 400 mL of free water, a good starting point*

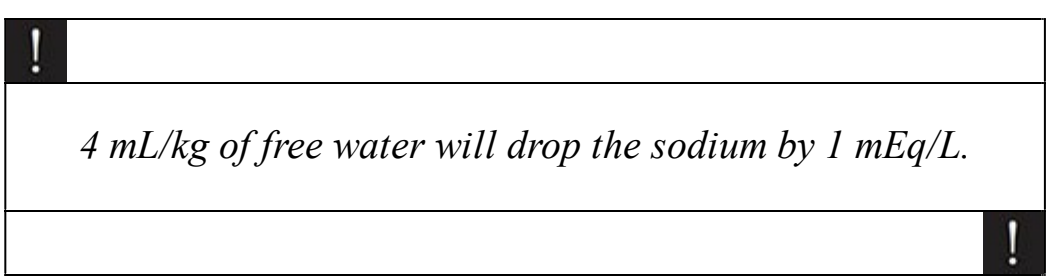
Table 8-2 shows how this formula is applied in a clinical scenario. Frequent measurement of the serum sodium is necessary to follow the gradual decrease in sodium levels and make appropriate fluid changes.

Table 8-2	SAMPLE CALCULATION FOR DETERMINING AMOUNT OF FREE WATER NECESSARY TO CORRECT SERUM SODIUM LEVEL

Case: A 6-month-old infant weighing 8 kg has serum sodium level of 157 mEq/L.
Free water deficit = (8 x 0.6) x 1 – (145/157) x (1000 mL/L)
365 mL = 4.8 x 0.076 (1000 mL/L)
Quick calculation: 4 mL x 8 kg x 12 mEq/L = 384 mL of free water.
Maintenance fluid amounts for an 8-kg child: (100 mL/kg x 8) = 800 mL/24 h.

a. Hypernatremia Due to Diabetes Insipidus

Diabetes insipidus (DI) is an uncommon condition that results in severe hypernatremia and excessive free water loss. It has either a CNS or nephrogenic (inherited or acquired) etiology. The diagnosis in the acute case is made by observing polyuria, a urine specific gravity <1.005, urine osmolality <200 mOsm/L, serum hypernatremia, and hyperosmolality (serum osmolality ≥295 mOsm/L).



The most common causes in the acute setting are CNS injuries, including severe head trauma and tumors (craniopharyngiomas). Elevated intracranial pressure can cause DI, and it is commonly seen in association with brain death. Nephrogenic DI is relatively rare, usually presents in a less acute setting, and is most commonly due to a hereditary defect. Treatment of DI consists of replacement of free water and administration of intranasal or IV desmopressin or vasopressin with strict monitoring of fluid balance and electrolytes.

B. Potassium

Potassium, the most abundant cation in intracellular fluid, is involved in many cellular processes, including protein synthesis. Maintenance of a transmembrane potential results in an electrical gradient across cell membranes. Changes in this resting membrane potential result in muscle excitation and nerve conduction. In particular, potassium plays a key role in myocardial contractility and function.

Potassium levels are tightly regulated by renal excretion – the main mechanism being distal tubular secretion; however, patients might develop hyperkalemia due to a combination of renal dysfunction and increased potassium release from injured tissue.

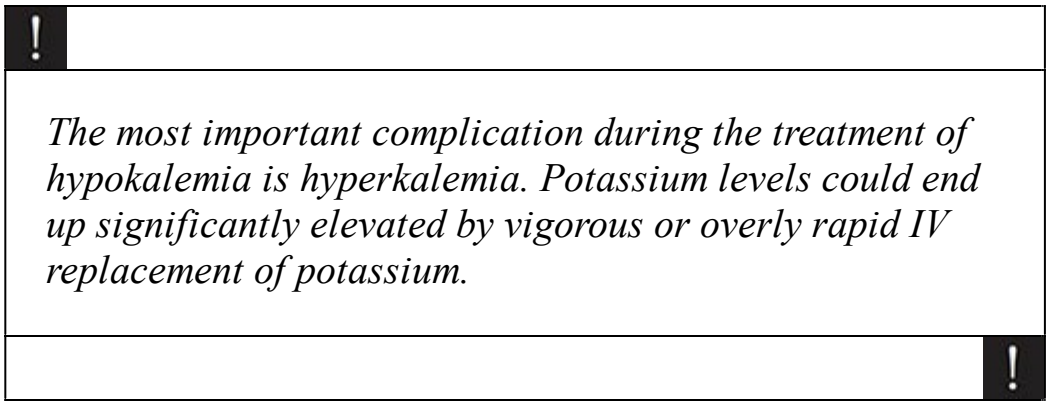
1. Hypokalemia

Hypokalemia, defined as a potassium level <3.5 mEq/L, may occur due to inadequate intake, renal losses, and gastrointestinal losses, following insulin therapy, and in association with metabolic alkalosis. Diuretics (especially furosemide and bumetanide), hyperventilation, mannitol, β-agonists (eg, frequent use of inhaled bronchodilators), and amphotericin B can cause hypokalemia.

Gastrointestinal losses via continuous nasogastric suctioning or diarrhea can also result in a significant drop in serum potassium levels.

Signs and symptoms include fatigue and paresthesias. Electrocardiogram (ECG) changes, including U waves and ventricular dysrhythmias (**Figure 8-2**), can occur when the potassium level drops below 2.5 mEq/L. Treatment of hypokalemia may include oral or IV supplementation, depending on the patient's status and the urgency of symptoms.

If the patient is receiving enteral feedings, the safest approach to correcting asymptomatic hypokalemia (no ECG changes) would be the gradual replacement of potassium by adding it to the dietary intake. Supplements (1- 3mEq/kg/d) can be administered enterally in 3 or 4 divided doses to correct hypokalemia or to prevent it in patients who are on diuretics without potassium-sparing properties. Potassium chloride can be an irritant to the gastric mucosa, might not be well tolerated, and may result in diarrhea.


!
The most important complication during the treatment of hypokalemia is hyperkalemia. Potassium levels could end up significantly elevated by vigorous or overly rapid IV replacement of potassium.
!

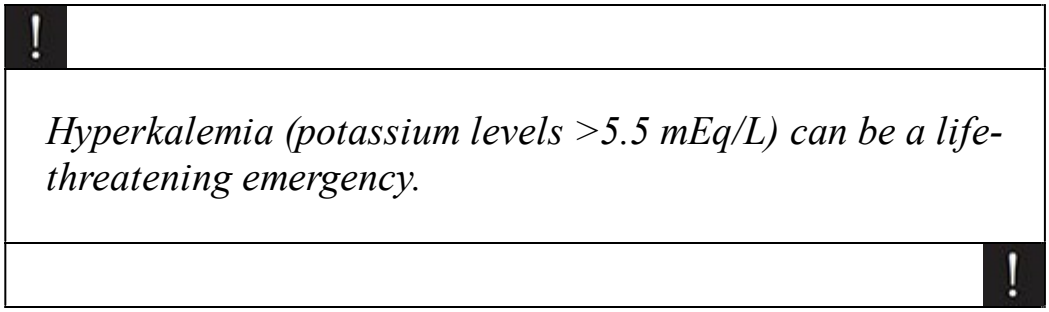
A conservative protocol for IV replacement is outlined below. These recommendations are considerably more stringent than those employed in the care of adults.

- For K^+ levels between 3.0 and 3.5 mEq/L, administer 0.25 mEq/kg IV KCl over 1 hour.
- For K^+ levels between 2.5 and 3.0 mEq/L, administer 0.5 mEq/kg IV KCl over 2 hours.
- For K^+ levels <2.5 mEq/L, administer 0.75 mEq/kg IV KCl over 3 hours. A potassium level should be checked halfway through this infusion.
- A safe alternative is to administer KCl in aliquots of no more than 0.5 mEq/kg/h, with a maximum dose of 10 mEq over 1 hour.
- A KCl infusion is recommended to be administered via a central venous or a large-bore IV line with adequate solution dilution and ECG monitoring whenever possible.

2. Hyperkalemia

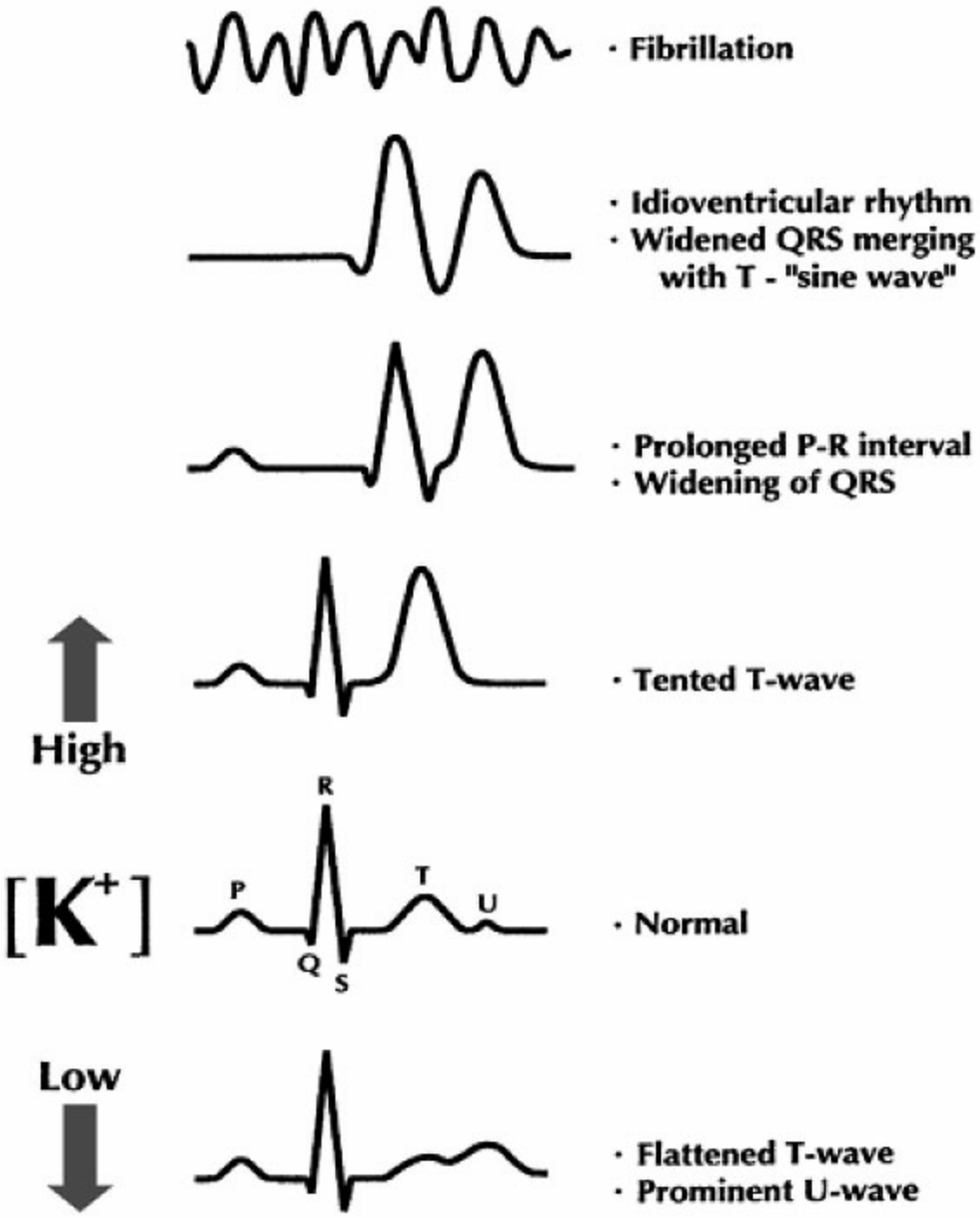
Hyperkalemia, or potassium level >5.5 mEq/L, requires urgent confirmation and rapid institution of therapy to lower the levels, especially if ECG changes are present. Since renal clearance of excess potassium is fairly efficient, hyperkalemia is unusual in the setting of adequate renal function. The condition can occur as a result of renal failure (acute or chronic), hypoaldosteronism, adrenal insufficiency, metabolic acidosis, muscle and tissue necrosis (rhabdomyolysis, burns, crush injuries),

tumor lysis syndrome (Chapter 17), and excessive intake or administration of potassium. Acidosis plays a role as well, increasing potassium levels by 0.5 mEq/L for every 0.1 decrease in the pH. Medications such as potassium-sparing diuretics (spironolactone), angiotensin-converting enzyme inhibitors, and digoxin can also lead to hyperkalemia, especially in the setting of concomitant renal dysfunction. Hemolysis from heel-stick blood sampling or tourniquet application is a common cause of a false elevation of potassium levels; however, it should not be assumed that this is the cause without demonstrating a normal potassium level on a repeated free-flowing sample without hemolysis.



Signs and symptoms of hyperkalemia are usually related to inadequate cardiac output due to arrhythmias. ECG changes include tall, peaked T waves, widening of the QRS complex, atrioventricular block, bradycardia, and eventually ventricular tachycardia or asystole (**Figure 8-2**). Weakness and paresthesias can occur when potassium levels exceed 7 mEq/L.

Figure 8-2. Illustrated Electrocardiographic Alterations with Hypokalemia and Hyperkalemia



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Symptomatic hyperkalemia requires simultaneous urgent intervention and therapy:

- Place the patient on a cardiorespiratory monitor with a rhythm strip and obtain a 12-lead ECG.
- Recheck the electrolytes to confirm the hyperkalemia.
- Discontinue any exogenous potassium being administered.
- Administer 1 or more of the following therapies:

– Calcium gluconate: 100 mg/kg given IV over 3 minutes (1 mL/kg of 10% solution) to stabilize the myocardium and prevent arrhythmias. Another dose may be repeated in 5 minutes if ECG

abnormalities persist.

- Sodium bicarbonate: 1 to 2 mEq/kg given IV over 10 to 15 minutes. Ensure the adequacy of ventilation prior to administration. Since calcium and bicarbonate will precipitate, the line must be flushed clear between these 2 medications.
- Insulin-glucose infusion to move potassium intracellularly: insulin 0.1 U/kg/h, mixed with 25% dextrose solution equivalent to 0.5 g/kg/h (2 mL/kg/h). The first hour's dose can even be administered rapidly (over 30 minutes) prior to initiating the continuous infusion. Glucose levels should be followed hourly for the duration of this infusion.
- Albuterol by multiple-dose inhaler or nebulizer in conjunction with 1 or more of the therapies outlined: albuterol is a weaker therapy and may not significantly change the potassium level, which is why it must be administered in conjunction with other therapies.
- An exchange resin, such as sodium polystyrene resin, administered 1 g/kg rectally. If the patient's hemodynamics and airway are stable, it can be administered orally or by nasogastric tube at the same dose (1 g/kg) every 6 hours. The rectal route has a rapid effect, whereas the oral/nasogastric route has a gradual and more sustained effect. This agent exchanges sodium for potassium along the gastrointestinal tract in an approximate ratio of 1 g/kg resin to 1 mEq/L decrease in K^+ level. Sodium levels should be monitored if more than 1 dose is used due to the potential for hypernatremia.

- Emergency hemodialysis: this should be added to the previous steps if the hyperkalemia is life-threatening and immediate correction is desired. Because it takes time to initiate this procedure, particularly if a large-bore indwelling line is required, all other modalities should be instituted while awaiting initiation of hemodialysis.

C. Magnesium

Magnesium is a cofactor for adenosine triphosphate-related functions, such as oxidative phosphorylation, protein synthesis, and DNA transcription. It is abundant in most diets and daily intake is usually adequate. Intracellular magnesium is responsible for most of the physiologic functions requiring adenosine triphosphate and magnesium. Normal plasma levels are 1.5 to 2.3 mg/dL (0.37-0.57 mmol/L).

1. Hypomagnesemia

Hypomagnesemia (<2 mg/dL) occurs fairly frequently (20%-50%) in ICU patients, likely because of inadequate or absent dietary intake combined with increased gastrointestinal and renal losses. Pancreatitis can result in hypomagnesemia and concomitant hypocalcemia as well. Patients with renal dysfunction are less likely to develop hypomagnesemia as they have limited ability to excrete magnesium in the urine.

Hypomagnesemia is most commonly associated with hypocalcemia and may result in the clinical features of hypocalcemia, such as neuromuscular weakness, muscle wasting, and ECG changes (increased PR interval, QT prolongation, and flat T waves). Hypokalemia is also associated with

hypomagnesemia, and correcting the magnesium levels might help in the management of persistent hypokalemia. Laryngospasm and seizures may also be seen in severe cases.

Causes of hypomagnesemia include:

- Nothing by mouth status and lack of IV supplementation
- Malabsorption due to familial hypomagnesemia, laxative use, short bowel syndrome
- Increased renal losses secondary to diuretic use, amphotericin B, chemotherapy, aminoglycosides, intrinsic renal disease (acute tubular necrosis)

Treatment of hypomagnesemia (symptomatic or asymptomatic patients with magnesium levels <1 mg/dL) is accomplished with slow infusions of magnesium sulfate, 25 to 50 mg/kg (2.5-5.0 mg/kg elemental magnesium), over 3 or 4 hours. Above a serum level of 2 mg/dL (0.82 mmol/L), the kidneys will waste the rest, so no additional benefit is gained from rapid IV replacement.

2. Hypermagnesemia

The kidneys usually clear excess magnesium, and hence hypermagnesemia is rarely encountered except in cases of renal dysfunction. Symptomatic hypermagnesemia is seen once levels rise above 4 mg/dL (>1.65 mmol/L).

Signs and symptoms include nausea, vomiting, decreased tendon reflexes, and neuromuscular blockade. Cardiovascular effects include bradycardia, myocardial depression, and ECG changes, including prolonged PR interval and atrioventricular block. Neonates may present with hypotonia and apnea.

Treatment consists of administering calcium gluconate (50-100 mg/kg IV), followed by restriction of further intake, hydration with normal saline, and administration of a loop diuretic.

D. Calcium

Calcium is a divalent cation, 99% of which is found in the skeleton; the remaining 1% is widely distributed throughout the body. Dietary sources of calcium include dairy products, seafood with bones, and green, leafy vegetables. Calcium is essential for teeth, bone mineralization, and muscle activity, especially myocardial excitation-contraction coupling. It also plays a pivotal role in the coagulation cascade. Data also suggest a role in cellular mechanisms of injury and necrosis. Excessive use of calcium during resuscitation and postresuscitation stabilization might be detrimental to the myocardium and other tissues (**Chapter 3**).

Because almost 50% of total calcium is in the active, ionized form and most of the remaining is bound to albumin and inactive, ionized calcium levels should be evaluated wherever possible. Levels are closely regulated by interplay of the parathyroid hormone, vitamin D, calcitonin, and renal excretion.

1. Hypocalcemia

Hypocalcemia is defined as total calcium <2.12 mmol/L (<8.5 mg/dL) or ionized calcium <1 mmol/L. Typically, calcium deficits occur as a result of low dietary intake and/or a lack of vitamin D or parathyroid hormone. The presentation of hypocalcemia is usually part of a more profound protein-calorie malnutrition, but it can occur as a stand-alone deficiency. It also can occur as part of pancreatitis, but this is less common in pediatric patients. The most common cause of hypocalcemia in infancy is congenital deficiencies of parathyroid hormone, as seen in conditions such as DiGeorge syndrome.

Hypocalcemia is a common occurrence in critically ill patients, many of whom also tend to be hypoalbuminemic. It is better to measure the ionized calcium levels rather than rely solely on total calcium.

Tetany, irritability, hyperreflexia, weakness and paresthesias, muscle fatigue, stridor, and laryngospasm are all common neuromuscular manifestations of hypocalcemia. Cardiovascular effects include hypotension, bradycardia, and arrhythmias.

a. Neonate/Infant

Calcium levels in a term neonate drop after the first 24 hours and are then maintained by the regulatory hormones. Neonatal hypocalcemia can be encountered in association with maternal diabetes mellitus, toxemia of pregnancy, and maternal hyperparathyroidism. It can present with irritability, tetany, or seizures.

Neonates with DiGeorge syndrome (developmental anomaly of third and fourth pharyngeal pouches) can present with symptomatic hypocalcemia. These patients might exhibit lesions such as mandibular hypoplasia, hypoparathyroidism, T-lymphocyte deficiency (thymic dysfunction), and congenital heart disease (atrial septal defect, ventricular septal defect, aortic arch anomalies, pulmonic stenosis, and truncus arteriosus).

Hypocalcemia can also occur in neonates as a result of hypomagnesemia. Correction of the hypomagnesemia may be necessary to correct the low calcium.

b. Child

Hypocalcemia in the older child is usually a result of a disruption of one or more of the regulatory mechanisms controlling calcium levels. Hypoparathyroidism, vitamin D deficiency, inadequate dietary intake, and renal losses are common conditions resulting in hypocalcemia, but the condition may also be seen in any critically ill or injured child.

Aggressive calcium supplementation is reserved for symptomatic and documented hypocalcemia. It is important to assess renal function and check other electrolytes, particularly magnesium levels, in the evaluation and management of hypocalcemia. More detailed evaluation for the cause of hypocalcemia requires tests of parathyroid function, renal function (phosphate levels), and levels of vitamin D metabolites (25-hydroxyvitamin D and 1,25-dihydroxyvitamin D).

c. Treatment

First, it is necessary to treat the cause of hypocalcemia. Therapy with IV calcium should ideally be

administered through large-bore or central venous lines since calcium extravasation can result in a chemical burn with consequent tissue necrosis. Do not administer calcium via scalp veins, intramuscularly, or subcutaneously.

Indications for IV calcium administration include hypocalcemia (ionized calcium levels <1 mmol/L [<4 mg/dL]), hyperkalemia, calcium channel blocker overdose, hypermagnesemia, and postarrest stabilization if there is laboratory evidence of hypocalcemia.

Calcium gluconate is the preferred calcium salt in neonates and may be administered orally or intravenously in the older infant and child. A 10% solution of calcium gluconate contains 0.45 mEq/mL of ionized calcium. The dosage is 50 to 200 mg/kg IV over 5 to 10 minutes for neonates, and 50 to 125 mg/kg IV over 5 to 10 minutes in infants and children. A continuous infusion may be administered (starting dose, 10–30 mg/kg/h) to maintain adequate calcium levels to prevent symptoms. The rate of the infusion can be titrated based on serial calcium measurements (or ionized calcium, if necessary).

Calcium chloride is the most readily bioavailable form of calcium. A 10% solution contains 1.36 mEq/mL of ionized calcium. It is administered in a dose of 10 to 20 mg/kg IV over 5 to 10 minutes through a central venous line. Rapid administration can result in bradycardia and hypotension.

The results of studies that compare calcium chloride to calcium gluconate show that they have similar bioavailability and are equally effective in correcting ionized hypocalcemia.

2. Hypercalcemia

Hypercalcemia, defined as a total calcium level >11 mg/dL (>2.75 mmol/L) or ionized calcium level >1.3 mmol/L, results from calcium release from bone. It can be seen following prolonged immobility, hyperparathyroidism, malignancies, excessive intake of vitamin A or D, and granulomatous disease.

Though relatively rare in pediatric patients, hypercalcemia can present with signs and symptoms that might lead to a wrong diagnosis. Due to the presence of hypertension and decreased level of consciousness, a patient with hypercalcemia could be mistaken for one with impending intracranial herniation. Other signs and symptoms are seen as cardiovascular and neuromuscular effects and include hypertension, shortened QT interval, irritability, lethargy, seizures, coma, nausea, vomiting, and abdominal pain.

The primary source of the hypercalcemia must be addressed and therapy directed specifically at lowering serum calcium levels. Acute hypercalcemia with total calcium levels >15 mg/dL requires aggressive attempts at lowering the levels.

Hydration with IV isotonic saline (200-250 mL/kg/d) in conjunction with furosemide-induced diuresis (1 mg/kg IV every 6 hours) provides a rapid response by increasing renal calcium excretion (calciuresis). Close monitoring of electrolytes, including phosphorus and magnesium, should be performed during diuresis.

Recombinant calcitonin has a rapid onset of action, acts by blocking bone resorption, and promotes calciuria. The dose of calcitonin is 10 U/kg IV, and it can be repeated every 4 to 6 hours. Mithramycin, aspirin, and indomethacin are alternative therapies that can be used to treat

hypercalcemia. Glucocorticoids can reduce calcium absorption from the gastrointestinal tract. Hydrocortisone, 1 mg/kg every 6 hours, is effective in curtailing calcium absorption but is less useful in acute hypercalcemia.

E. Phosphate

Adequate quantities of phosphorus are achieved with a regular diet. In the body, phosphorus is present as phosphate salts and is closely associated with calcium intake. Phosphate levels are regulated by the renal functions of filtration and proximal renal tubular reabsorption.

The main effects of phosphate occur in membrane phospholipids, bone, adenosine triphosphate, and 2,3-diphosphoglycerate. These four systems alone make phosphate homeostasis critical to many basic functions in the body.

1. Hypophosphatemia

Decreased intake and excessive excretion of phosphorus occur classically in diabetic ketoacidosis. Insulin therapy further reduces phosphate levels by driving it into the cells. Renal dysfunction, such as acute tubular necrosis, renal tubular acidosis, Wilson disease, hypokalemia, and hyperparathyroidism all can lead to hypophosphatemia. Patients receiving IV fluids alone (without phosphorus supplementation), those ingesting aluminum-containing antacids, and the critically ill can become hypophosphatemic.

Signs and symptoms of hypophosphatemia in the acute setting include muscle weakness, hypoventilation, myocardial dysfunction, seizures, and coma. Treatment consists of the addition of phosphate (as sodium and potassium salts) to the IV fluids when serum phosphorus levels are below 1 mg/dL (0.323 mmol/L). Sodium phosphate contains 3 mmol (94 mg) PO_4 and 4 mEq/mL sodium; potassium phosphate contains 3 mmol (94 mg) PO_4 and 4.4 mEq/mL potassium. Replacement is given as 0.16 to 0.32 mmol/kg IV over 4 to 6 hours, and either salt can be used. If renal dysfunction coexists and potassium phosphate is used, potassium levels must be monitored closely. Also, phosphate infusions or supplements should not be administered in the presence of hypercalcemia.

2. Hyperphosphatemia

Excessive intake (rare) and reduced excretion, such as in renal failure, hypoparathyroidism, pseudohypoparathyroidism, tumor lysis syndrome, and rhabdomyolysis, may result in hyperphosphatemia. A hemolyzed blood sample can deliver an erroneous level indicating hyperphosphatemia.

The main effect of hyperphosphatemia is the occurrence of hypocalcemia due to chelation when the total calcium measurement is multiplied by the inorganic phosphorus product >60 mg/dL. Treatment includes aluminum hydroxide antacids enterally and rehydration with isotonic fluids. Hypocalcemia should be treated aggressively as well.

III. METABOLIC DISEASE

A. Glucose

Glucose is essential for tissue metabolism, and its levels are maintained within a tight range by the actions of insulin. Disorders of glucose metabolism can lead to hypoglycemia and hyperglycemia with fairly significant clinical consequences.

1. Hypoglycemia

Hypoglycemia is defined as a serum glucose level <60 mg/dL (<3.3 mmol/L). Weakness, sweating, tachycardia, tremors, and eventually seizures might develop with hypoglycemia. The CNS symptoms are particularly evident because the brain is dependent on a steady source of glucose for neural activity. Symptoms are varied, particularly in the neonate.

Hyperinsulinism, inborn errors of metabolism, endocrine disorders (including adrenal and pituitary dysfunction), ketotic hypoglycemia, and drugs and toxins – including exogenous administration of insulin – are the broad categories of conditions leading to hypoglycemia. An extensive endocrine and metabolic workup is necessary to establish the diagnosis. Propranolol and other β-blockers might mask the symptoms (tachycardia, sweating) of hypoglycemia. Laboratory tests, including cortisol, insulin, growth hormone, urine ketones, serum chemistry, and liver function tests, should be obtained during the hypoglycemic episode, if possible.

Once hypoglycemia has been confirmed, immediate treatment with IV glucose should be initiated. Administration of 2 to 4 mL/kg of 25% dextrose IV will provide 0.5 to 1 g/kg of glucose. This solution can be diluted 1:1 so that a 12.5% solution is injected into a peripheral vein or intraosseously. Because dextrose is fairly hyperosmolar, neonates should not be given more than 12.5% dextrose. A bolus of 10 mL/kg of 5% dextrose will provide 0.5 g/kg of glucose.

2. Hyperglycemia

A blood glucose level >150 mg/dL is considered hyperglycemia. This condition is not unusual in the critically ill child. It is the result of a stress response and the presence of high levels of circulating epinephrine and other anti-insulin stress hormones. Glucose levels should be monitored in the postresuscitation stabilization period.

B. Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA), a diagnosis sometimes missed, can lead to cerebral edema with catastrophic neurologic outcomes. A newly diagnosed diabetic child (approximately 30% present in DKA) or a known insulin-dependent diabetic patient can present with this condition and requires prompt attention to fluid management and correction of the acidosis.

An intercurrent illness might trigger the episode of DKA. Patients present with headaches, emesis,

abdominal pain, lethargy, and clinical signs of dehydration. There may be a history of weight loss, polydipsia, and polyuria. The abdominal pain and emesis might lead to a mistaken diagnosis of an intra-abdominal process or "surgical abdomen." If the dehydration is severe, evidence of hypovolemic shock may be seen at presentation. The classic clinical signs of rapid/Kussmaul respirations have been mistaken for asthma and respiratory distress. The presence of a fruity (ketotic) odor is helpful in making the diagnosis.

DKA is defined as a blood glucose concentration >200 mg/dL, with ketonemia/ketonuria, and a venous pH <7.3 or bicarbonate <15 mEq/L. The severity of DKA is categorized on the basis of the degree of acidosis (mild, venous pH <7.3 or bicarbonate <15 mEq/L; moderate, pH <7.2 or bicarbonate <10 mEq/L; severe, pH <7.1 or bicarbonate <5 mEq/L).

1. Initial Stabilization

Once initial laboratory studies confirm the diagnosis, the mainstay of therapy is the administration of insulin by continuous infusion and correction of the dehydration. If the history is suggestive of an infectious trigger, this should be investigated and appropriately treated.

An initial fluid bolus of 10 mL/kg of normal saline is administered, with an additional bolus given if the patient is hemodynamically unstable as a result of the dehydration. Only isotonic fluid should be administered during this initial fluid resuscitation phase. Lactated Ringer solution should be avoided if renal function is significantly impaired.

An insulin infusion—without a bolus of IV insulin—should be initiated at 0.05 to 0.1 U/kg/h. Subsequent IV fluids can be started using normal saline with 20 mEq/L of potassium chloride and 20 mEq/L of potassium phosphate (as long as urine output is adequate) at 1.5 to 2 times maintenance, for a total amount not to exceed 3,500 mL/m²/d. A dextrose solution (10% dextrose in normal saline with potassium chloride and potassium phosphate) can be added once the blood glucose level is below 300 mg/dL.

Levels should be brought down by no more than 100 mg/dL (5.5 mmol/L) every hour. A more rapid decline might require the addition of dextrose to the IV fluid sooner, even if the serum level remains elevated above 300 mg/dL. Because insulin is responsible for correction of the acidosis, the insulin infusion should be maintained while adjusting the dextrose solution as required to effect an appropriate adjustment in blood glucose level.

Administration of sodium bicarbonate is not indicated unless severe acidosis, with resultant hemodynamic compromise or arrhythmias from hyperkalemia, is present. Sodium bicarbonate is potentially harmful as it causes significant osmolar shifts and potentially worsens cerebral edema.

Hourly monitoring of blood glucose and blood gases (every 2-4 hours), frequent monitoring of electrolytes (including sodium, potassium, calcium, magnesium, and phosphate), and cardiovascular ECG monitoring should be performed.

2. Cerebral Edema

Most patients with DKA might have some degree of cerebral edema at presentation. The main risk factors for progression are administration of large quantities of hypotonic fluids, rapid hourly declines in blood glucose levels, and hyponatremia. The latter commonly occurs in DKA and is also a result of the hyperglycemia and hypertriglyceridemia (pseudohyponatremia).

$$Na^+ \text{ corrected} = Na^+ \text{ observed} + [(\text{glucose in mg/dL} - 100/100) \times 1.6]$$

If both hyperglycemia and hypertriglyceridemia are fully corrected, the sodium level can be predicted using the following formula:

$$\{\text{Reported sodium (mEq/L)}\}\ \{0.021[\text{triglycerides (g/dL)} + 0.994] + 0.016\ [\text{glucose (mg/dL)}]\}$$

C. Adrenal Insufficiency

1. Primary Adrenal Insufficiency

Congenital adrenal hyperplasia is often seen in the first 2 or 3 weeks of life, presenting as shock with associated hyponatremia, hypoglycemia, and hyperkalemia. It is characterized by a group of disorders that affect mineralocorticoids and glucocorticoid production due to enzymatic deficiency in the pathways leading to the synthesis of cortisol from cholesterol. The high levels of corticotropin lead to hyperplasia of the adrenal glands. Typically, neonates with or without ambiguous genitalia will present in shock out of proportion to the duration of symptoms. They have elevated levels of steroid metabolites. Partial enzyme deficiencies may also occur and require a corticotropin stimulation test for diagnosis.

2. Tertiary Adrenal Insufficiency

This is the most common form of adrenal insufficiency seen in the pediatric ICU. Patients present with a history of weakness, emesis, abdominal pain, and fever. Tertiary adrenal insufficiency may occur because the patient is on long-term steroid therapy (such as for asthma), resulting in the suppression of the hypothalamic-pituitary-adrenal axis. An intercurrent illness might result in the patient presenting with shock out of proportion to the duration or severity of symptoms. Adrenal insufficiency should be suspected in the presence of hypoglycemia, hyponatremia, hyperkalemia, and metabolic acidosis together with azotemia. The presence of a random cortisol level of <5 μg/dL (137 mmol/L) in a stressed, critically ill patient is highly suggestive of adrenal suppression or insufficiency. The corticotropin and cortisol levels are low in tertiary adrenal insufficiency.

Increasingly, adrenal insufficiency is being diagnosed early in a variety of critically ill children. It should be considered in any infant or child who remains hypotensive despite adequate volume resuscitation and initiation of appropriate vasoactive agents (catecholamine-resistant shock). A

random cortisol level should be obtained. An initial dose of 2 mg/kg IV hydrocortisone may be administered and can be continued at 1 mg/kg every 6 hours if the cortisol level in a profoundly stressed patient remains below 25 μg/dL.

Alternative treatments use dexamethasone (0.2 mg/kg IV every 6 hours) or methylprednisolone (1 mg/kg IV every 6 hours). Dehydration and hypoglycemia are treated with adequate fluid resuscitation with normal saline boluses and the correction of glucose levels.

Key Points

Fluids, Electrolytes, and Neuroendocrine Metabolic Derangements

- Conditions affecting the sodium balance include SIADH, cerebral salt wasting, and diabetes insipidus.
- Acute adrenal insufficiency occurs in congenital adrenal hyperplasia (neonates) or as a tertiary condition in patients with long-term steroid use. It presents with hypovolemia (shock), hypoglycemia, hyponatremia, hyperkalemia, and azotemia.
- In a neonate with hyponatremia, hyperkalemia, hypoglycemia, and acidosis, a diagnosis of congenital adrenal hyperplasia must be considered and management started immediately.
- Sodium levels should be corrected slowly (change of 0.5-1.0 mEq/L/h). A rapid drop can lead to cerebral edema, and rapid increase can lead to central pontine myelinolysis.
- Hyperkalemia with associated ECG changes requires immediate therapy.
- Hypotonic maintenance fluids should be avoided in pediatric patients, especially those at risk of SIADH.
- The potassium in IV fluids should be adjusted to the needs of a patient's urine output and potassium levels. Avoid the standard use of 20 mEq/L of potassium chloride in IV fluids in all patients.
- Rapid IV fluid resuscitation with isotonic fluid (normal saline or Ringer lactate) should be accompanied by frequent reassessment of clinical status.
- In the management of DKA, blood glucose levels should not be dropped >100 mg/dL/h.
- An initial IV bolus of insulin is not indicated in the treatment of DKA.
- Sodium bicarbonate should not be used to correct metabolic acidosis in DKA patients.

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TRAUMATIC INJURIES IN CHILDREN

Objectives

- Describe how children's patterns of injury differ from those of adults.
- Outline priorities for the management of the injured child.
- Employ life-saving therapies in the injured child.

I. INTRODUCTION

Trauma remains the leading cause of morbidity and mortality in children and adolescents, whose injury mechanisms and patterns can differ significantly from those of adults. Accidental and abusive injuries account for a significant proportion of emergency department visits and use of healthcare resources.

II. PRIMARY SURVEY

Evaluation of an injured child begins with a rapid and systematically performed primary survey, following the *ABCDE* checklist with the goal of identifying and intervening in immediately life-threatening problems:

Airway and cervical spine: evaluation and management of the airway (with spinal motion restriction)

Breathing: assessment of breathing and provision of supplemental oxygen or assisted respirations

Circulation: determining circulatory adequacy, identifying and controlling external hemorrhage

Disability: rapidly assessing neurologic status, with attention to level of consciousness, evidence of traumatic brain injury, focal deficits, and checking for hypoglycemia

Exposure (temperature control): fully exposing and examining the patient while supporting body temperature

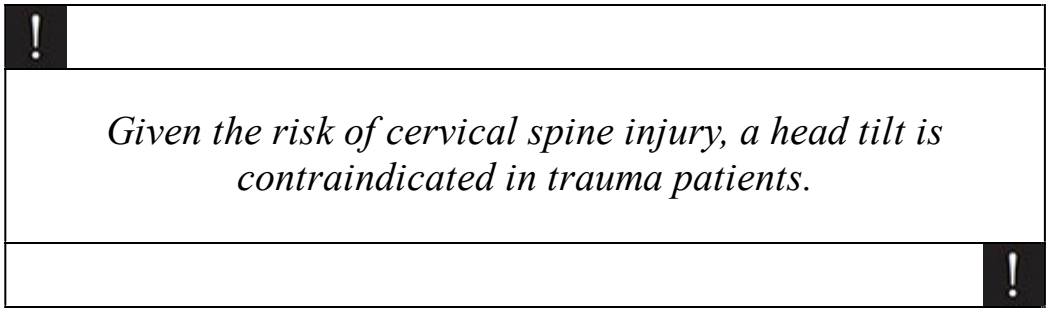
The team should not move on to the next step until any life-threatening issues have been addressed (ie, if the airway is obstructed, it must be opened before assessing breathing; if oxygen is needed, it is started before checking for circulatory adequacy). There are two caveats to this. First, tension

pneumothorax should be decompressed before tracheal intubation and positive pressure ventilation are instituted. Second, massive external hemorrhage, which will be rapidly lethal unless controlled; it should be managed by external compression and, if necessary, placement of a commercially available manufactured arterial tourniquet and/or use of hemostatic dressings before the airway and cervical spine assessment. With multiple providers present, one can deliver treatment while another continues the assessment.

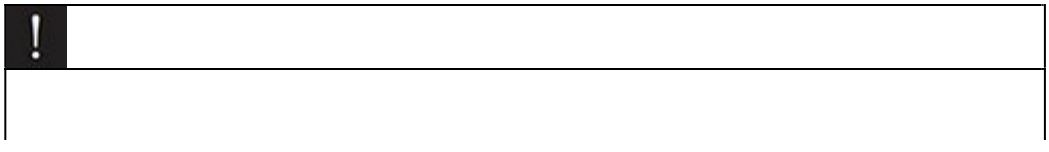
Motion of the cervical, thoracic, and lumbar spine should be restricted prior to moving a trauma patient. Typically, the patient is placed in a semirigid collar and on a long spine board or similar device at the event scene. Before placement of the collar, in-line, bimanual, spinal motion restriction should be assured. If a collar is not placed at the scene, one should be applied as soon as possible. Infants and young children placed on the long spine board should have a 1-inch thick layer of padding beneath them from shoulders to hips. This helps maintain a neutral cervical spine position and minimizes the development of pressure ulcers.

A. Airway

Airway evaluation begins with an assessment of airway patency with the patient in the neutral position. In infants and young children, this is accomplished using a 1-inch thick layer of padding under the shoulders. In older children, this layer should be placed beneath the entire body, including the head. This difference is due to the infant's proportionally larger head size and prominent occiput, and a propensity to neck flexion when supine. If airway patency cannot be achieved by proper positioning, several maneuvers may be used, including a jaw thrust. Head tilt must be avoided to forestall potential reinjury to the cervical spinal cord. Every effort should be made to recognize airway compromise promptly and secure a definitive airway given the potential for progressive airway loss.



Airway adjuncts may also be helpful in displacing the tongue and pharyngeal tissue from the airway. An oropharyngeal airway should be used only in an unconscious patient as it may cause significant gagging and vomiting. The length of the airway is determined by measuring the distance from the corner of the mouth to the angle of the mandible. A nasopharyngeal airway may be used in a conscious, cooperative patient. The length of this airway is determined by measuring from the nares to the tragus. Laryngeal mask airways (LMAs) are also useful, especially when an endotracheal tube (ETT) cannot be quickly placed, and are sized based on the patient's weight.



Placement of nasopharyngeal airways should be avoided in patients with midface trauma, basilar skull trauma, evidence of cerebrospinal fluid rhinorrhea, or severe coagulopathy.
Determination of correct endotracheal tube size:
1) Broselow Pediatric Emergency Tape
2) (age/4) + 4 (uncuffed) or 3.5 (for cuffed tubes)

Endotracheal intubation is indicated when a trauma patient is unable to maintain an airway because of obstruction or trauma, loss of airway tone, respiratory failure or decompensated shock, or severe neurologic dysfunction (Glasgow Coma Scale [GCS] score ≤8). It is also indicated if a patient has a potential inhalational injury or requires airway protection from aspiration of gastric contents or blood. Though traditionally, uncuffed endotracheal tubes have been used for patients younger than 8 years, cuffed tubes are prefered if properly sized and kept deflated or appropriately inflated. Patients at risk for impaired gas exchange, such as those with acute respiratory distress syndrome, inhalation injury, or pulmonary contusions, should be intubated with a cuffed endotracheal tube regardless of age because they may require high ventilatory pressures to achieve adequate oxygenation and ventilation.

All trauma patients are assumed to have a full stomach. Endotracheal intubation should be performed using a rapid-sequence technique to minimize risk of emesis and aspiration. Hypotension with administration of medications for intubation should be anticipated and measures taken to avoid this (eg, careful selection of sedatives and administration of volume). The use of ketamine for anesthesia induction and sedation in trauma has the advantage of maintaining the patient's blood pressure, given that it causes a release of endogenous catecholamines. Though traditionally ketamine was to be avoided in traumatic brain injury, recent reviews suggest that it is safe and may be advantageous. Succinylcholine is contraindicated in cases of hyperkalemia, neuromuscular disease, and burns.

All essential supplies must be gathered before intubation. A helpful pneumonic for this is SOAP ME:

S: Suction: ideally a Yankauer suction catheter with suction turned on

O: Oxygen: adjusted to the proper flow rate

A: Airway supplies: properly sized ETT, a smaller size in case of airway swelling, stylet, and airway adjuncts (including LMA)

P: Pharmacy: medications for anesthesia induction and emergency medications

M: Monitors: basic cardiorespiratory monitors, including end tidal CO_2, and stethoscope

E: Emergency equipment: including the code cart

Oral endotracheal tube depth insertion formula: (age/2) +

12, or 3 times size of ETT.

Ideally, the most skilled individual places the ETT given the complications that trauma adds, including difficult visualization due to C-spine precautions, potential for blood and loose teeth in oropharynx, and risk for aspiration from a full stomach.

B. Breathing

Airway patency alone does not ensure adequate ventilation and oxygenation, so breathing should be evaluated. The goal is normal oxygenation and ventilation. Pulse oximetry should be employed with a goal of maintaining a saturation of at least 94%. Oxygen may be provided by nasal cannula, face mask, non-rebreather face mask, LMA, or ETT. Ventilation may be provided by a bag-mask device or a mechanical ventilator, with a goal of a normal Pco_2. In cases of suspected head injury, hyperventilation will decrease cerebral perfusion and potentially cause secondary injury; however, controlled hyperventilation (ie, to a Pco_2 – not end-tidal CO_2 – of 35 mm Hg) should be employed in cases of impending cerebral herniation to decrease cerebral blood flow and lower intracranial pressure (ICP). The use of continuous capnography (end-tidal CO_2) is recommended to monitor ventilation and ensure tube placement, especially during patient transport. Patients with signs of tension pneumothorax require immediate needle decompression, followed promptly by tube thoracostomy. Some providers perform "finger thoracostomy" to avoid iatrogenic pneumothorax. Patients with signs of open pneumothorax (sucking chest wound) require an occlusive dressing followed by tube thoracostomy. The timing of the tube thoracostomy may depend upon other urgent treatments and evaluations.

C. Circulation

Circulatory compromise with trauma in children can result from many different injuries. Blood volume, cardiac output, and bleeding must be considered. Hemorrhagic shock is the most frequently encountered form of shock in trauma patients. Children may lose up to 30% of their circulating blood volume secondary to hemorrhage and still maintain normal blood pressure, so hypotension is a very late sign of pediatric shock. As such, hypoperfusion must be aggressively treated before frank hypotension develops. Applying direct pressure to actively bleeding open wounds is vital to control external hemorrhage when applicable. A commercially manufactured arterial tourniquet may be used to control hemorrhage from a bleeding extremity if direct pressure fails. Hemostatic dressings and sealants have also been successfully utilized by the military, and may have potential application for severe bleeding in children.

Other types of shock that may be seen in trauma victims include obstructive shock resulting from tension pneumothorax or cardiac tamponade, neurogenic shock resulting from spinal cord injury, and less commonly, cardiogenic shock resulting from trauma to the myocardium or valvular rupture.

Patients with signs of tension pneumothorax require immediate decompression, followed by tube thoracostomy. Placement of an invasive airway, or any positive pressure breaths, should always be delayed until after initial decompression in the setting of a tension pneumothorax. Those with signs of hemorrhagic or neurogenic shock need volume resuscitation. Intravenous access should be obtained peripherally. After 2 unsuccessful attempts (or 90 seconds duration) by a practitioner experienced at gaining IV access, an intraosseous needle should be placed, preferably in the proximal tibia. Central venous catheterization should not be performed outside of the operating room or pediatric ICU.

Though commonly administered, crystalloid fluids are frequently overutilized in pediatric in trauma, and there is mounting evidence of harm in the form of increased coagulopathy, risk of compartment syndromes, and multiorgan failure. In the case of hemorrhage, blood products should be utilized as soon as possible, ideally via a massive transfusion protocol. With hemorrhagic shock, 10 mL/kg of packed red blood cells should be administered. Should further transfusion be required, and/or in the presence of evidence of continued hemorrhage, packed red blood cells should be given together with plasma and platelet concentrates in a volume ratio of 1:1:1 or 1:1:2 (plasma: platelet concentrate: packed red cells). A platelet concentrate unit is considered one-sixth of an apheresis platelet unit, so that a single apheresis unit is given for every 6 units of packed red cells and plasma.

Severely injured patients can present with trauma-induced coagulopathy, which has been shown to be associated with an increased mortality. This coagulopathy can be made worse if not aggressively addressed with blood products (plasma and platelets), avoiding excessive crystalloid or colloid administration, and treating and preventing hypothermia and acidosis. Cryoprecipitate, or fibrinogen concentrate, should be administered if the fibrinogen level falls below 150 mg/dL at a dose of one unit for every 10kg body weight. Hypocalcemia and hypomagnesemia can occur during a massive transfusion using citrated blood products, so appropriate monitoring and correction are needed. Many centers are utilizing viscoelastic testing to monitor coagulation and fibrinolysis; thromboelastography (TEG) or rotational thromboelastometry (ROTEM) can give quick results and help guide hemostatic therapy. There is also emerging evidence of the benefit of tranexamic acid (TXA), an antifibrinolytic, if given within 3 hours of injury; suggested dosing is 15 mg/kg (maximum, 1 g) followed by 2 mg/kg/h for 8 hours. Finally, rigorous thermoregulation to prevent hypothermia is mandatory, and pH should be maintained as close to normal as possible.

Neurogenic shock may be present if the spinal cord is injured above the sixth thoracic vertebra. After 60 mL/kg of volume has been administered, consider vasopressor medications (norepinephrine or phenylephrine) if the patient continues to remain hemodynamically unstable.

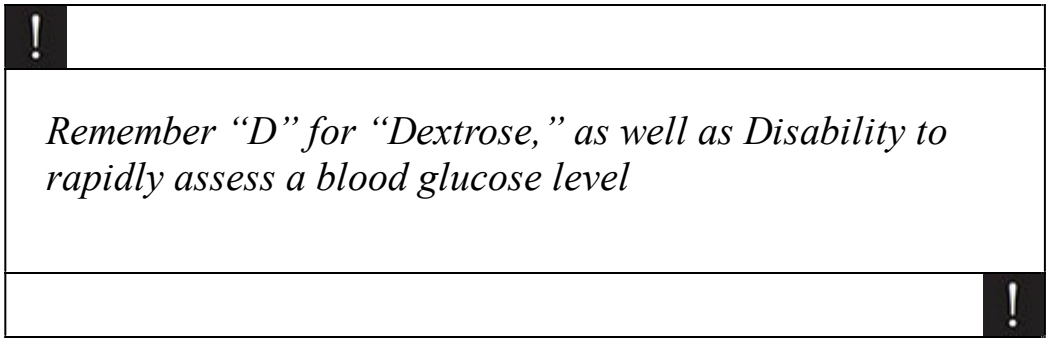
Cardiac tamponade may be present in the setting of a narrowed pulse pressure, muffled heart sounds, hypotension, and distended neck veins, ultimately leading to a pulseless electrical activity and traumatic cardiac arrest. This can be confirmed on ultrasound and requires immediate operative decompression and repair of the associated cardiac wound. Pericardiocentesis may be needed as a temporizing measure if a qualified surgeon is not immediately available.

In all cases of shock, a urinary catheter should be placed to monitor urine output and adequacy of resuscitation. Urine output reflects adequacy of renal perfusion, and therefore cardiac output. The urethral meatus should be inspected first for bleeding, as this could indicate urethral disruption and would be a contraindication to Foley catheter placement requiring a urology consult. Inadequate intravascular volume and preload will be reflected by inadequate urine output. Ongoing hemodynamic

instability may require operative intervention for definitive hemorrhage control.

D. Disability

Following stabilization of airway, breathing and circulation, a neurologic assessment (for "disability") should be performed. Assessment should include the level of consciousness, pupillary response, and detection of any lateralizing or localizing signs, paraplegia, and/or paralysis. The GCS score should be tabulated during the primary survey, again during the secondary survey, and frequently thereafter. This score, used as a predictor of neurologic outcome, ranges from 3 (worst) to 15 (best). The components of the score are motor function (1-6 points), verbal response (1-5 points), and eye opening (1-4 points). Elements of the verbal score are modified to account for developmental differences in young children and infants (**Chapter 15, Table 15-1**). Any abnormality in neurologic status should prompt expeditious neurosurgical evaluation if available. Finally, hypoglycemia may worsen neurological outcome. Therefore, a rapid blood glucose determination is essential in all critically ill or injured patients upon presentation to the emergency department.



E. Exposure

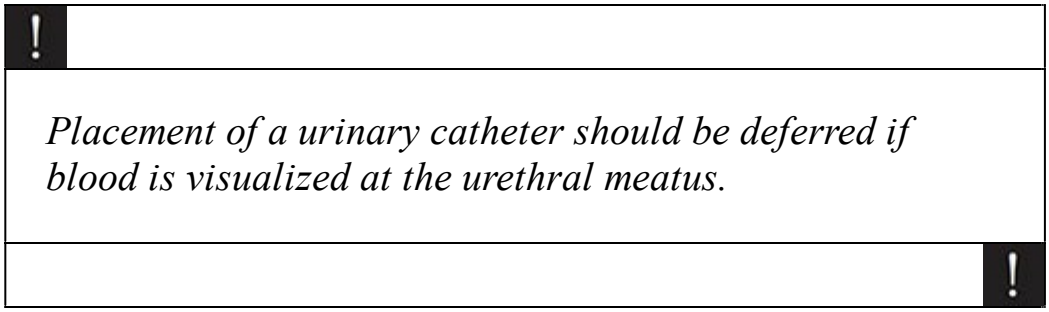
Pediatric trauma patients should be exposed by removing all clothing, log-rolling the child, and removing the backboard to allow examination of the back for life-threatening injuries. Patients should then be covered with warm blankets to prevent hypothermia, which can exacerbate coagulopathy (by impairing platelet function and coagulation factor action) and worsen hemodynamic instability. The room temperature should be warmed as should IV resuscitative fluids.

F. Adjuncts to the Primary Survey

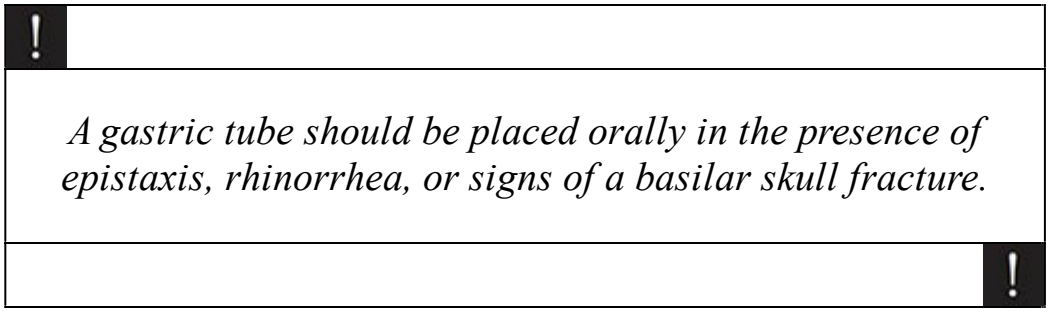
Depending on the nature of a patient's trauma, a number of additional tests, procedures, and devices may be used as adjuncts to the primary survey, including the following:

- Cardiopulmonary monitoring
- Pulse oximetry
- Blood pressure monitoring
- End-tidal CO_2 monitoring

- Blood gas analysis
- Placement of a urinary catheter to measure output and assess renal perfusion
- Placement of a gastric tube to decompress the stomach and prevent aspiration
- Extended-focused assessment for sonography in trauma (E-FAST) scan and radiography

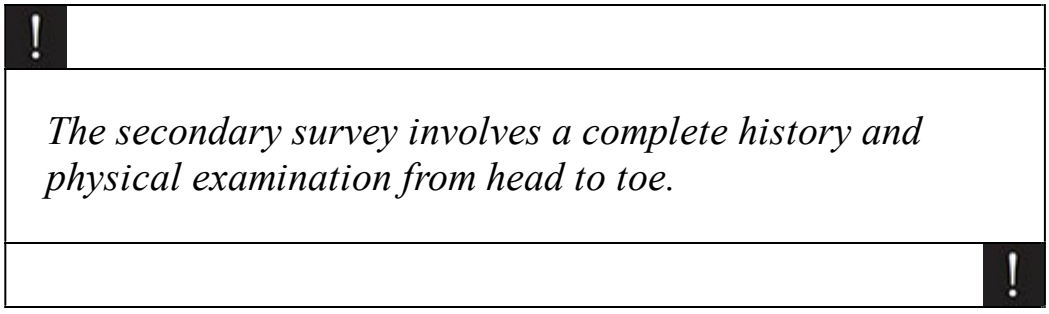


Portable radiographs of the chest, pelvis, and (circumstances permitting) the lateral cervical spine should be obtained during the primary survey. Such radiographs may identify injuries that require immediate attention. The E-FAST scan is another essential primary survey adjunct. Using ultrasound, the following areas can be quickly assessed for the presence of potential bleeding: perihepatic, perisplenic, pericardium, and pelvis. The extended scan also includes assessing both thoracic fields for hemo- and pneumothoraces.



III. THE SECONDARY SURVEY

The secondary survey is done when the primary survey has been completed, resuscitative efforts are underway, and the patient is stable. In unstable patients requiring aggressive resuscitative measures, this may be delayed. The purpose of the secondary survey is to find less obvious or physiologically disturbing injuries, or conditions threatening to patient survival if not found and treated in the next few hours.

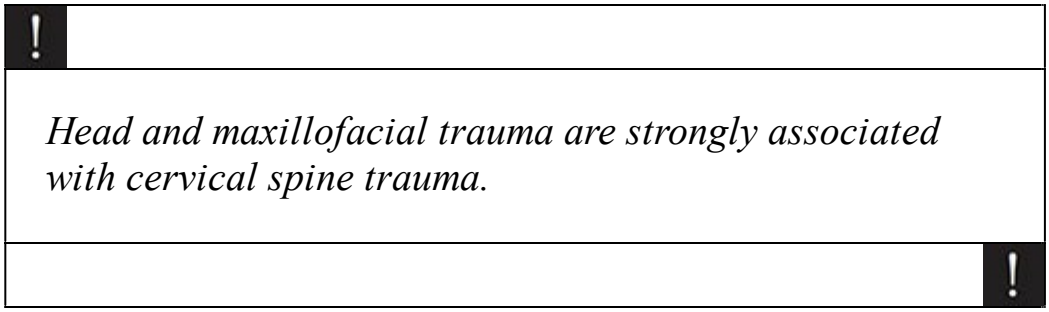


A. History

The mnemonic for important components of the trauma history is SAMPLE: symptoms, allergies, medications, past medical history (and pregnancy status for girls of childbearing potential), last meal or liquids consumed, and events and environment surrounding the injury. Knowing the mechanism of injury is helpful because it predicts the pattern of injuries likely to be identified. In addition, all medications and alternative preparations to which the child has been exposed should be reviewed and reconciled in the medical record.

B. Physical Examination

The physical examination should proceed from head to toe. In an infant, the anterior fontanelle should be evaluated. The head of the patient's bed is elevated by 30° (if not contraindicated) and the fontanelle is palpated. A full or bulging fontanelle may be a sign of increased ICP. The head is examined for lacerations and significant irregularities indicating fractures. Raccoon eyes (periorbital ecchymosis) and the Battle sign (ecchymosis overlying the mastoid process) suggest a basilar skull fracture. The eyes are examined for pupillary response, subconjunctival hemorrhages, and extraocular movements. The nose is examined for epistaxis, rhinorrhea (suggesting leakage of cerebrospinal fluid [CSF]), and fractures. The oropharynx is evaluated for lacerations and injury to the teeth.



In the secondary survey of the neck, the cervical collar is removed and in-line, bimanual, spinal motion restriction is temporarily applied. The neck and cervical spine are palpated for muscle spasm, vertebral step-offs, and other deformities, tenderness, and crepitus. The carotid pulses should be assessed, and the position of the trachea should be evaluated. Following the neck examination, the cervical collar is replaced. The chest is inspected for penetrating injuries and associated sucking chest wounds, as well as deformity or obvious fracture, then palpated to determine the presence of tenderness or crepitus. The lungs are auscultated for equality and clarity of breath sounds. Inequality of breath sounds may occur due to pneumothorax or hemothorax. The heart is examined with attention to the rate, rhythm, and quality of heart sounds. Distant, muffled heart sounds may represent cardiac tamponade, which may be accompanied by tachycardia with a narrow pulse pressure. The abdomen is inspected for ecchymoses, auscultated for the presence and quality of bowel sounds, and palpated for tenderness, peritoneal irritation, and rigidity.

If the backboard was not removed during the primary survey, it is removed during the secondary survey. The patient is log-rolled off the backboard, and his/her back is inspected for vertebral step-offs, other deformities, ecchymoses and palpated for tenderness. Every effort should be made to remove the backboard within 2 hours to prevent the development of pressure ulcers. Patients should

be log-rolled every 2 hours thereafter to prevent pressure ulcers until the spine is cleared. The genitalia are inspected for lacerations and blood at the urethral meatus; a rectal examination is performed to assess tone and detect the presence of blood in the rectal vault. The musculoskeletal system is inspected and palpated, and peripheral pulses evaluated, to identify fractures, dislocations, and physical signs suggesting compartment syndrome.

The patient's level of consciousness and pupillary response should be reassessed in the secondary survey. The GCS score should be tabulated again. Reflexes should be assessed, and the presence of any sensory and motor deficits should be determined. Spinal cord injury should be localized by determining the level of motor and sensory function (**Tables 9-1** and **9-2**). If present, deficits are best recorded on the American Spinal Injury Association spinal injury chart.

Table 9-1 MOTOR LEVELS

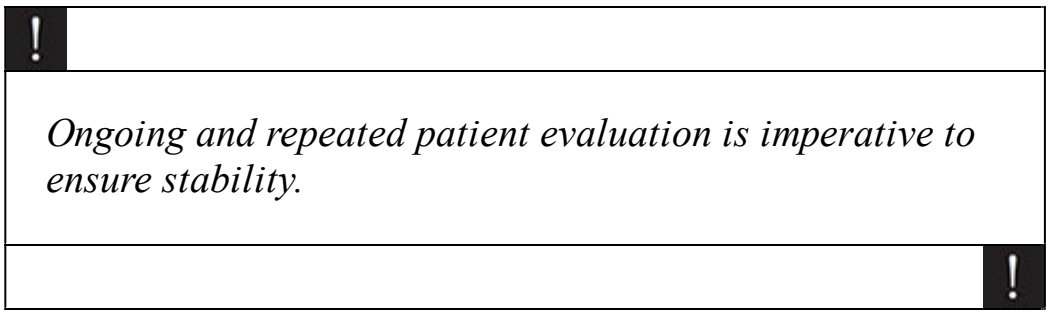
Nerve Root	Major Muscles Affected
C3-5	Diaphragm
C5	Elbow flexors
C6	Wrist extensors
C7	Elbow extensors
C8	Finger flexors
T1	Finger abductor (fifth finger)
L2	Hip flexors
L3	Knee extensors
L4	Ankle dorsiflexors
L5	Long toe extensors
S1	Ankle plantar flexors

Table 9-2 SENSORY LEVELS

Nerve Root	Major Sensory Area Affected
C4	Clavicle
C6	Thumb
C7	Second and third fingers
C8	Fifth finger
T4	Nipples
T10	Umbilicus
L1	Inguinal ligament
L3	Lower anterior thigh/knees
L5	Big toe
S1	Lateral foot
S3-5	Perineum

C. Adjuncts to the Secondary Survey

The results of the secondary survey may point to the need for additional studies. These may include hematologic and biochemical tests, computed tomography (CT) scans, and completion of the cervical spine radiographs. Obtain head and/or abdominal CT according to the clinical decision rules established by the Pediatric Emergency Care Applied Research Network (PECARN) to avoid excessive radiation exposure and minimize the possibility of late cancers. If there are concerns for neck injury, or the physical exam or radiographs are inconclusive, consider a neck CT or magnetic resonance imaging with surgical consultation based on the local protocol. Additional radiographs of the thoracolumbar spine and extremities, angiography, and ultrasonography may also be indicated.



From a practical standpoint, labs are often obtained during the primary survey. In any major injury, serial hematocrit measurements are important to monitor for bleeding from a solid organ injury, such as a splenic, hepatic, or renal laceration. A rising white blood cell count or amylase and lipase levels may be an important clue to an evolving bowel or pancreatic injury. Hepatic transaminases can provide early indication of blunt injury to the liver. Urinalysis will help exclude renal contusions or lacerations. Given that anemia from blood loss and clotting dysfunction from consumptive coagulopathy commonly occur in the critically injured patient, blood samples for typing and crossmatching should be sent to the laboratory with these initial studies, so that blood products can be given as soon as they are required. Serial blood gas measurements are also vitally important during the stabilization of any severely injured ventilated child, as they will direct ventilatory support. Base deficit values can guide resuscitation efforts.

IV. MANAGEMENT OF TRAUMATIC INJURIES

A. Traumatic Brain Injury

Case Study

A 5-year-old boy was hit by a car in a parking lot when running in between rows. He was knocked unconscious for approximately 30 seconds, followed by a period where he was moaning and crying.

When emergency medical services arrived, he opened his eyes on command, followed motor commands, and continued crying but was not speaking. He was immobilized with placement of a semirigid cervical collar and long spine board before transport. On arrival to the emergency department, he cries for his mother, opens his eyes to voice, and localizes to pain. Vital signs reveal a temperature of 97.8°F (36°C), a heart rate of 66 beats/min, a respiratory rate of 24 breaths/min, and a blood pressure of 140/67 mm Hg. Oxygen saturation on pulse oximetry (Spo_2) is 90% on room air. Physical examination demonstrates a sluggishly reactive right pupil, briskly reactive left pupil, and large right cephalohematoma. Subsequently the boy becomes more lethargic, opening his eyes only to voice, making incomprehensible sounds, and withdrawing from pain. His right pupil is now slightly dilated. A head CT scan shows a large right biconvex extradural (epidural) mass lesion associated with midline shift and ventricular effacement, and an adjacent nondisplaced right parietal skull fracture.

Detection

– What are the next steps in managing this patient?

– Did this patient exhibit a classic lucid interval?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need to be intubated or receive other therapeutic interventions?

Effective Communication

– With this change in the patient's clinical status, who needs the information and how will it be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How should the treatment strategy be implemented?

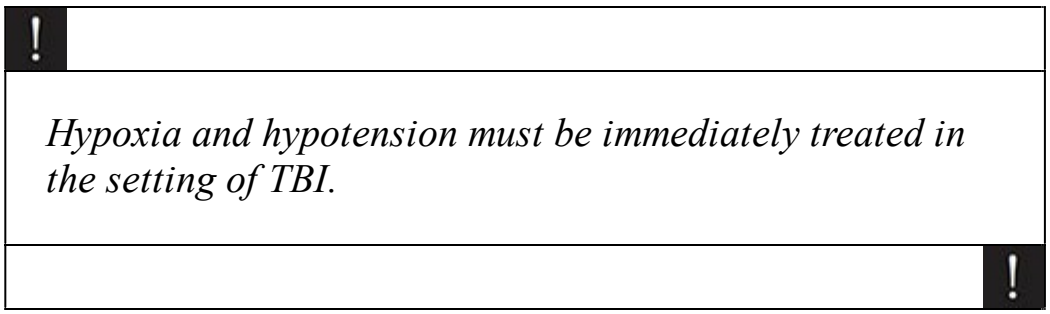
– Who is to do what and when?

Traumatic brain injury (TBI) contributes to a significant number of trauma deaths in children and usually occurs in motor vehicle collisions, falls, bicycle accidents, and sports injuries. Nonaccidental trauma remains a significant cause of brain injuries in children younger than 2 years. TBI may be characterized as mild (GCS score 13-15), moderate (GCS score 9-12), or severe (GCS score 3-8).

Children with mild brain injury generally do well, but as many as 53% have radiographic abnormalities on head CT despite a lack of significant findings on physical examination. Up to 57%

of children and 85% of infants with epidural hematomas have no apparent loss of consciousness at the time of impact, and 7% have no alteration of mental status at any time following the injury. These statistics underscore the likelihood that a life-threatening injury may exist despite a benign presentation. Children with a normal neurologic examination and a normal head CT at presentation rarely deteriorate and can be safely discharged home. Fortunately, the recently established PECARN clinical prediction rule for brain injury has shown that head CT and its attendant late cancer risks can be safely avoided in children 2 years and older who have normal mental status, no loss of consciousness, no vomiting, nonsevere injury mechanism, no signs of basilar skull fracture, and no severe headache. This is also true in children younger than 2 years who have normal mental status, no scalp hematoma, no loss of consciousness or loss of consciousness for less than 5 seconds, nonsevere injury mechanism, no palpable skull fracture, and who are acting normally according to the parents.

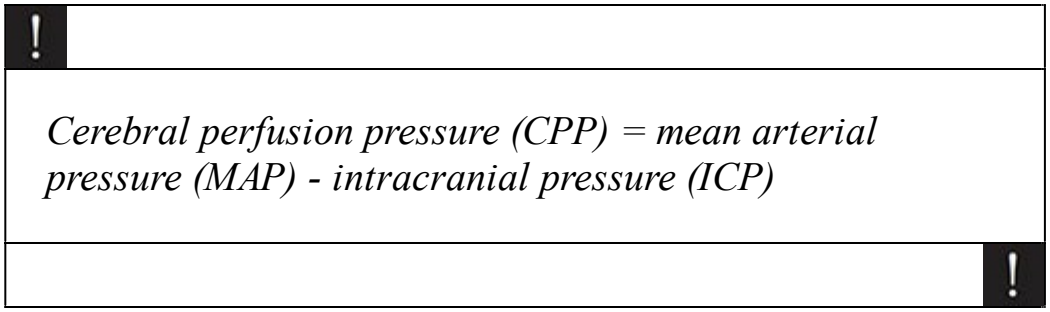
Most patients with moderate to severe TBI will not present with intracranial mass lesions but rather with diffuse cerebral swelling due to a combination of mostly vasogenic edema at the locus of injury and hyperemia elsewhere, resulting from loss of cerebral arteriolar pressure autoregulation. Such patients are managed chiefly by nonsurgical means, although drainage of CSF via ventriculostomy may be required to assist in lowering the ICP. However, intracranial bleeding with mass effect may require prompt neurosurgical evaluation, followed by neurosurgical intervention for drainage or decompression. Epidural hematomas, while uncommon in childhood, are associated with a good prognosis if promptly recognized and neurosurgically evacuated. However, as previously stated, the classic lucid interval typically observed in adults with this condition is most often absent in children. Subdural hematomas, by contrast, are both more common and far more deadly, owing to the associated cerebral tissue injury that is invariably present. Neurosurgical evacuation is indicated if a midline shift exceeds 5 mm, or if a large intracerebral hematoma is contributing to the mass effect.



The management of children with moderate to severe TBI requires careful attention to ventilator and hemodynamic status as well as to ICP. The goal is to prevent the primary brain injury from being adversely affected by the hypoxia and hypoperfusion that may result from inadequate resuscitation, termed *secondary brain injury*. Children with moderate TBI (GCS score 9-12) may therefore require intubation and ventilation for indications other than the TBI itself. Intubation, ventilation, and ICP monitoring are indicated in those with a severe TBI (GCS score 3-8), and should be strongly considered in head-injured patients whose GCS score deteriorates rapidly by ≥2 points. When a patient with head trauma meets this criterion, the goals of mechanical ventilation are normal oxygenation (Spo_2 94%-99%) and normal ventilation ($Paco_2$ 35-40 mm Hg [4.7-5.3 kPa]), since both hypercarbia and hypocarbia may worsen outcome by contributing to hyperemic or ischemic injury. Thus, hyperventilation is indicated only in cases of impending or actual cerebral herniation. Meticulous attention to respiratory and hemodynamic status is imperative, because hypoxemia and hypotension have each been shown to significantly increase morbidity and mortality in patients with

TBI. Control of seizures and maintenance of normal body temperature (avoidance of fever) are additionally of key importance in inhibiting the progression of brain injury to cell death and encephalomalacia. Adequate analgesia and sedation must also be provided. The head of the patient's bed should be elevated by 30°, if not otherwise contraindicated, and the child's head should be positioned in the midline, avoiding flexion, abduction, rotation, and any constricting ties or dressings that could impair jugular venous return.

In patients with severe TBI (GCS score 3-8), an ICP monitor may be placed in the lateral ventricle (ventriculostomy), subdural space (bolt), or brain parenchyma. Ventriculostomy catheters may be preferred because they allow both monitoring of ICP and drainage of CSF as a means of lowering ICP. Normal ICP in adults is <10 mm Hg, lower in young children and infants. Abnormal ICP exceeds 20 mm Hg at any age. Acute sustained increases (spikes) in ICP are promptly treated with sedation, analgesia, and mild hyperventilation to lower CO_2. Hypertonic saline (3%) infusions (5-10 mL/kg) may be administered as an osmotic agent for increased ICP. If 3% saline is not available, IV mannitol, 0.5 g/kg (range 0.25-1 g/kg), can be used to raise serum osmolality (300-320 mOsm/L) and lower ICP. Other modalities utilized for control of ICP include sedation, neuromuscular blockade, elevation of the head, maintenance of normothermia, strict avoidance of hyperthermia requiring active cooling modalities, and use of endotracheal or IV lidocaine before suctioning. Seizures must be ruled out and, if present, managed aggressively.



Cerebral perfusion pressure (CPP) must be monitored closely in patients with TBI. In adults, a CPP of 60 to 70 mm Hg is usually targeted. There are no data documenting the appropriate CPP in children and infants. However, given their lower MAP and ICP readings, general ranges can be extrapolated. A reasonable range is 50 to 60 mm Hg in children and 40 to 50 mm Hg in infants. While volume can be administered to improve CPP, the goal should be to restore euvolemia, as hypervolemia can exacerbate cerebral edema.

As noted, hypertonic saline solution (3%) is preferred for control of continuous elevations of ICP, because it increases serum osmolality without causing intravascular volume depletion. A sodium of 150-155 mmol/L is generally targeted; however, this fluid has very high chloride content (513 mmol/L), which may exacerbate acidosis and renal injury. In cases of intractable elevations of ICP and low CPP with euvolemia, inotropic or vasopressor agents should be used to optimize CPP by increasing MAP, even to the point of relative systemic hypertension. The use of mannitol to decrease cerebral edema as well as ICP has fallen out of favor except in cases of intractable intracranial hypertension, because mannitol is a potent osmotic diuretic that may cause hypotension. Moreover, ICP may rebound after its discontinuation. The use of corticosteroids has no role in TBI and may be harmful. Based on several recent studies, therapeutic hypothermia is contraindicated and may increase mortality. For refractory elevated increased ICP, high dose barbiturates may be considered.

Decompressive craniectomy may lower ICP, but is associated with worse long-term outcomes.

B. Spinal Cord Injury

Case Study

A 15-year-old boy presents to the emergency department after diving into the shallow end of the pool. The lifeguard witnessed the accident, noting that he struck his forehead causing a hyperextension of his neck. The lifeguard immediately responded and found that the boy was unable to move his lower extremities and had significant weakness of the upper extremities. He had no loss of consciousness and no evidence of aspiration. His vital signs were stable in the field, and he was immobilized with a semirigid collar and long spine board before transport. In the emergency department, the patient is awake, alert, and afebrile, with a respiratory rate of 9 breaths/min, oxygen saturation of 94% on room air, heart rate of 56 beats/min, and blood pressure of 70/40 mm Hg.

Detection

– What is the first step in stabilizing this patient?

– Why is this patient hypotensive?

– What is the diagnosis?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Are other therapeutic interventions indicated to augment blood pressure?

– Are other therapeutic interventions indicated to augment ventilation?

Effective Communication

– Who needs to know about this patient's clinical status, and how will the information be communicated?

Teamwork

– How should the treatment strategy be implemented?

– Who is to do what and when?

Stretching of the spinal cord can result in spinal cord injury without radiographic abnormality (SCIWORA) in children but not adults. An absence of radiographic abnormality cannot exclude complete injury.

Cervical spine injury is uncommon in children. It has a mortality rate of 15% to 20%, largely due to its association with significant brain injury. Young children (<11 years) most frequently sustain cervical spine injury as the result of motor vehicle collisions, falls, and pedestrian accidents. They are at risk for high cervical injuries, cervical spine dislocations, and spinal cord injury without radiographic abnormality (SCIWORA). The latter is due to the horizontal orientation of the facet joints and the elastic nature of the intervertebral ligaments, which permit upper cervical spinal elements to shift rather than break upon application of forces, leading to angular momentum of the head and neck. Older children (>11 years) and adults are at risk for low cervical injuries and cervical spine fractures. Children ages 10 to 11 years begin to show adult patterns of injury, and their transition to those patterns is eventually completed in late adolescence.

Differences in injury patterns may be attributed to anatomical differences between the young child and the older child/adult. Young children have proportionally larger heads with relatively weak neck muscles, which make them more susceptible to flexion and extension injuries. The vertebral bodies are incompletely ossified, and the intervertebral ligaments are far more elastic than those of adults. In addition, the vertebral bodies of the upper cervical spine are anteriorly wedged in children, predisposing them to forward vertebral movement and anterior dislocation, a condition termed *pseudosubluxation*. The fulcrum for cervical motion is at C2 to C3 in the young child compared to C5 to C6 in adolescents and adults. The young child's spinal cord may be easily stretched, torn, or sustain contusion secondary to laxity of spinal ligaments. The cord may stretch up to 5 cm without rupturing, whereas the adult cord may rupture after only 5 to 6 mm of traction.

Children with cervical and high thoracic spinal cord injuries (SCIs) are predisposed both to ventilatory compromise and neurogenic shock. Ventilatory compromise may occur due to denervation of the intercostal muscles associated with SCIs below C4, or denervation of the diaphragm and intercostal muscles associated with SCIs above C4. Neurogenic shock results from loss of sympathetic vascular tone by disruption of the sympathetic chains at T6 or above, leading to seemingly paradoxical bradycardia despite the presence of hypotensive shock. However, even in patients with SCI, hemorrhagic shock is far more common than neurogenic shock and may coexist with neurogenic shock. As such, a careful search for sources of bleeding should be made even if SCI is present, particularly in the first 1 or 2 hours.

Respiratory failure may require intubation for diaphragmatic paralysis, pulmonary toilet, or both. Neurogenic shock is treated with aggressive volume resuscitation, followed by selective use of vasopressor agents if hypotension persists despite adequate volume resuscitation. The term *spinal shock*, which refers to the truly paradoxical loss of spinal reflexes following injury, is sometimes

incorrectly used to describe the hemodynamic consequences of SCI, and use of this term should be limited to the neurologic sequelae. Finally, great care must be taken to prevent the development of pressure ulcers, which can begin within 1 hour after SCI. There is no scientific evidence supporting the use of corticosteroids in SCI; in fact, their use is associated with increased complications, such as hyperglycemia and avascular necrosis at large joints such as the hip.

The lateral cervical spine radiograph is often used as a screening test for CSI, but it will miss 15% of all fractures. The addition of anteroposterior and odontoid views will capture most other fractures. While CT is increasingly used for definitive diagnosis of cervical spine fractures in children and adults, its use is best limited to cases in which diagnosis by standard radiograph is equivocal and sensory or motor deficits are present, because of the associated risks of late cancers. A valid clinical prediction rule for cervical spine imaging following major trauma in children has not been developed, but 8 factors are known to be associated with it: altered mental status, focal neurologic findings, neck pain, torticollis, substantial torso injury, conditions predisposing to CSI, diving, and high-risk motor vehicle crash. Absence of such factors may militate against the use of routine CT in the diagnosis of possible CSI. Regardless, cervical spine precautions, including a semirigid collar and thoracolumbar spinal motion restriction, must be implemented and maintained until CSI is excluded by appropriate clinical and radiological means, including spine surgery consultation and magnetic resonance imaging, when indicated.

C. Thoracic Trauma

Case Study

A 4-year-old girl presents to the emergency department after sustaining a gunshot wound in the right lower chest in a drive-by shooting, apparently an unintended victim caught in the crossfire. Vital signs reveal a temperature of 98.4°F (37°C), a heart rate of 150 beats/min, a respiratory rate of 50 breaths/min, and a blood pressure of 70/35 mm Hg. Spo_2 is 88% on room air. There is a "sucking sound" coming from her anterior wound.

Detection

– What is the source of this patient's hypotension?

– How will you treat her?

Interventions

– What are the most immediate treatment strategies?

Reassessment

– Is the current treatment strategy effective?

– Will she require intubation? What should be done prior to intubation?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will this information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How should the treatment strategy be implemented?

– Who is to do what and when?

Thoracic injuries, which often occur as a result of blunt trauma, are the second leading cause of death due to trauma in children (after head injuries). The overall mortality rate from thoracic trauma has been reported to be 5%, increasing to 25% when accompanied by head and abdominal trauma. In infants and toddlers, thoracic injuries occur most commonly in the contexts of motor vehicle collisions and maltreatment. School-aged children are most likely to sustain thoracic trauma due to bicycle, scooter, and skating accidents, while teenagers are most commonly injured in motor vehicle collisions. While penetrating thoracic trauma is less common, the priorities of management remain the same. Occlusive dressings should be applied to open chest wounds, followed by tube thoracostomy.

A chest radiograph should be the initial diagnostic test in the evaluation of thoracic trauma. Due to their extremely compliant chest walls, children sustain rib fractures much less frequently than adults do and are at higher risk of pulmonary contusions. Therefore, if rib fractures are present, they signify an especially forceful impact. Pulmonary contusions are the most common thoracic injuries in children and may be associated with hypoxemia, hypoventilation, ventilation-perfusion mismatch, increased work of breathing, and decreased pulmonary compliance. There may be consolidation, edema, and alveolar hemorrhage in the area of contusion. Contusions may not be visible on early radiographs but will become apparent later. A high index of suspicion for thoracic trauma is indicated, given an appropriate mechanism of injury. Treatment includes avoidance of fluid overload, supplemental oxygen, analgesia, incentive spirometry, and mechanical ventilation if required (**Table 9-3**).

The presence of rib fractures, particularly posterior rib fractures, should raise suspicion of physical abuse. These fractures usually occur as the result of significant compression of the chest, as happens in shaking. Other potential etiologies include birth trauma and disorders of bone ossification, such as osteogenesis imperfecta and rickets, as well as the major forces transmitted from high impact injuries (ie, high speed motor vehicle crashes, falls from significant heights). Fractures of the first rib are associated with an especially high incidence of intrathoracic injuries, including pneumothorax and hemothorax, and should prompt CT angiography for diagnosis of significant vascular injury.

Table 9-3	DIAGNOSTIC EVALUATION
Type of Trauma	**Diagnostic Study**

Any	Serial hematocrit readings Complete blood cell count Prothrombin time/partial prothrombin time/fibrinogen Viscoelastic testing Complete metabolic panel, including aspartate aminotransferase/alanine aminotransferase, amylase, lipase Blood gas measurements Urinalysis Radiographs of the chest, cervical spine (posteroanterior, lateral, odontoid), and pelvis
Head	Head CT MRI: not indicated immediately; obtain later to aid in prognostication
Cervical spine	Cervical spine radiographs: posteroanterior, lateral, odontoid views Cervical spine CT: if plain radiographs are inadequate MRI: indicated when radiographs/CT scans are negative for bony injury but neck pain/tenderness is elicited; evaluates ligamentous injury
Thoracic	Chest radiograph Chest CT Chest CT angiography: indicated when serious vascular injury is suspected Electrocardiogram Echocardiogram: indicated when pericardial effusion is suspected Esophagogram: indicated when esophageal rupture is suspected Bronchoscopy/bronchography: indicated when injury to tracheobronchial tree is suspected
Abdominal	Aspartate aminotransferase/alanine aminotransferase Amylase/lipase Urinalysis FAST examination Abdominal CT with oral and IV contrast

CT, computed tomography; MRI, magnetic resonance imaging; FAST, focused abdominal sonography for trauma

Multiple rib fractures have been shown to be associated with severe injury in children; mortality rates rise as the number of fractured ribs increases. Flail chest occurs when ≥2 adjacent ribs are fractured in 2 places each, resulting in paradoxical chest wall movement (inward movement with inspiration and outward movement with exhalation), although muscle spasm may limit the motion of small flail segments.

One-third of traumatic pneumothoraces occur in isolation, the other two-thirds in association with other injuries. Often pneumothoraces are asymptomatic and only detected by chest radiography. Treatment is the evacuation of air with a tube thoracostomy. Accumulation of a large amount of air in the pleural space may lead to the collapse of the ipsilateral lung, with tracheal and mediastinal shift resulting in hemodynamic instability (tension pneumothorax). Signs of tension pneumothorax include tracheal shift away from the affected lung, absence of breath sounds and hyper-resonance to percussion on the ipsilateral side, tachycardia, hypotension, dyspnea, and hypoxemia. When clinically suspected, evacuation of tension pneumothorax must not be delayed by radiographic confirmation. Prompt decompression of tension pneumothorax via needle thoracostomy, followed by tube thoracostomy, is necessary to prevent hemodynamic collapse. Hemothorax can develop in the context of blunt or penetrating injuries to the chest with disruption of the thoracic vessels, intercostal blood vessels, or lung parenchyma. Evacuation of significant hemothorax is important in the prevention of subsequent infection, chronic atelectasis, and lung entrapment; acutely, hemothorax may compromise cardiac output due to both blood loss and obstructive shock. Ventilation-perfusion abnormalities are possible, but evacuation should be preceded by aggressive volume resuscitation.

Flail chest is often managed conservatively with analgesia but may require positive pressure.

Cardiac contusions most frequently occur as a result of blunt trauma and may present as chest pain, dysrhythmia, or hypotension. An electrocardiogram may show ST-segment changes, atrial tachycardia, sinus tachycardia, or premature beats. Treatment is generally supportive, with cardiopulmonary monitoring required only if abnormalities appear on the electrocardiogram. Cardiac tamponade may occur when blood accumulates in the pericardial space. Patients present with tachycardia, muffled heart sounds, and a narrow pulse pressure. The diagnosis is suspected when a penetrating injury is found within an area bounded by the nipple line, midclavicular lines, and costal margins, and is confirmed by bedside ultrasound or echocardiography. Treatment consists of fluid removal by pericardial window or emergency thoracotomy, although pericardiocentesis may be employed as a temporizing measure until a qualified surgeon performs evacuation, followed by identification and treatment of the bleeding source.

Diaphragmatic injuries are uncommon, but when they occur, they most frequently involve the posterolateral left hemidiaphragm. Upward coiling of a nasogastric tube and herniation of the abdominal contents into the thorax may be visualized on the chest radiograph. Up to 50% of these injuries are missed at the initial presentation, becoming apparent only on subsequent chest radiographs.

Esophageal injury is caused most frequently by penetrating injury. Blunt trauma may also induce a tear at the gastroesophageal junction due to acute movement of gastric contents into the esophagus. Gastric contents may spill into the pleural space or mediastinum. Diagnosis is made when extravasation of contrast material is seen on an esophagogram. Treatment includes antibiotics and surgical repair.

Injuries to the tracheobronchial tree are rare in children but are associated with a mortality rate of 30%. They may be caused by blunt or penetrating trauma. Patients present with subcutaneous emphysema, pneumothorax, hemoptysis, and persistent air leak following tube thoracostomy. Diagnosis may be confirmed by bronchoscopy, but bronchography may be required to visualize tears in the distal bronchial tree.

D. Abdominal Trauma

Case Study

An 18-month-old girl is brought to the emergency department by her babysitter. The girl is lethargic and difficult to arouse. There is no history of fever, illness, ingestion, or trauma. Vital signs reveal a

temperature of 96°F (36°C), a heart rate of 170 beats/min, a respiratory rate of 45 breaths/min, and a blood pressure of 64/40 mm Hg. Spo_2 is 95% on room air. After stabilization and removal of the child's clothing, a distended abdomen and several small bruises on her posterior thorax are discovered.

Detection

– What interventions would be needed to stabilize this patient?

– What is an appropriate diagnostic evaluation for this patient?

Intervention

– What are the most immediate treatment strategies?

– Should the patient be intubated?

Reassessment

– Is the current treatment strategy effective?

– Does she require additional fluid or blood products?

– Is a surgical consultation indicated?

– Are there findings suggestive of nonaccidental trauma?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will this information be disseminated?

– Where is the best place to manage the care of this child?

Teamwork

– How should the treatment strategy be implemented?

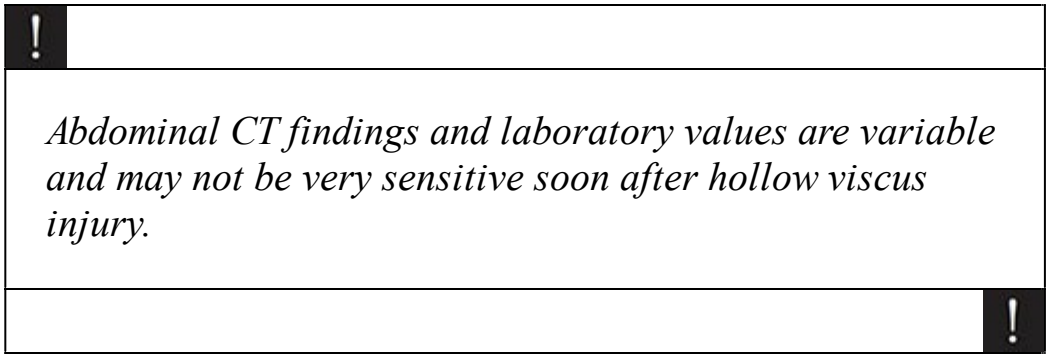
– Who is to do what and when?

Due to their proportionally larger solid organs and decreased abdominal wall musculature and fat, children are at higher risk than adults for sustaining significant organ injury as a result of abdominal trauma. Multiple organs may be injured, because an impacting force will strike a relatively large proportion of a child's body surface. Intra-abdominal injuries related to bicycle handlebars and poorly fitting car lap belts have a high likelihood of requiring surgical intervention. Physical findings include abdominal distention, abrasions, contusions, lap belt pattern ecchymoses, peritonitis, and focal or diffuse tenderness. Hemodynamically unstable patients require emergent resuscitation and laparotomy, whereas stable patients warrant further assessment.

Diagnostic assessment of abdominal trauma continues with a laboratory evaluation. Decreasing hemoglobin and hematocrit measures should raise suspicion of abdominal hemorrhage. Several

studies have shown that elevations in the serum aspartate aminotransferase and alanine aminotransferase levels may be associated with abdominal trauma. One study found that a serum alanine aminotransferase measurement above 131 U/L with abdominal tenderness is predictive of abdominal injury with a sensitivity of 100%. Serum amylase and lipase levels are variable predictors of pancreatic injury. Serum amylase may be normal when obtained soon after pancreatic injury, but will always be elevated ≥3 hours following injury. Elevations in serum amylase may also be seen in the presence of head injury and trauma to the salivary glands without pancreatic injury; subtype analysis to determine the source is possible but not routinely used in this context.

The focused assessment of sonography in trauma (FAST) exam is of some utility in the pediatric population and can be accomplished quickly by those who are skilled. Most solid abdominal organ injuries in hemodynamically stable children are managed nonoperatively. The FAST examination may be useful in two specific situations in the pediatric population: (1) in multiply injured, hemodynamically unstable patients, it may be used to detect abdominal hemorrhage as the source of instability; and (2) with the laboratory evaluation and physical examination, it may serve as a screening tool to identify patients who would benefit from CT.


!
Abdominal CT findings and laboratory values are variable and may not be very sensitive soon after hollow viscus injury.
!

Indications for abdominal CT include abnormal physical examination, abnormal laboratory values, lack of a reliable physical examination due to distracting injuries or altered mental status, or a compelling need for precise information regarding specific organ injury or grade of injury. Abdominal CT is the diagnostic procedure of choice for evaluation of abdominal injury and is most useful in detecting solid organ injuries, particularly those of the liver, spleen, and kidneys. It should be performed with IV contrast to best delineate these injuries. Pancreatic and intestinal injuries may be missed on initial abdominal CT, particularly if it is performed soon after injury. Oral contrast may be helpful on later scans if clinical signs suggest an occult small bowel injury. Again, the recently established PECARN clinical prediction rule for intra-abdominal injury requiring acute intervention has shown that abdominal CT and its late cancer risks can be safely avoided in children with no complaints of abdominal pain, no history of vomiting, no evidence of abdominal wall trauma (including seat belt sign), a GCS score ≥14, no abdominal tenderness, no evidence of thoracic wall trauma, and no decreased breath sounds.

Splenic injury is the most common injury associated with blunt abdominal trauma. Abdominal CT with intravenous contrast is its most specific detection method. Preservation of the spleen is the standard of care in children in order to prevent immunologic compromise. Most splenic injuries are therefore managed nonoperatively, reserving surgical intervention only for those patients with otherwise unmanageble hemodynamic instability. Unlike in adults, a contrast blush on CT is not by itself an indication for operation or embolization. Splenectomy is indicated only in cases of

hemodynamic instability with irreparable splenic damage.

Liver injury, the second most common injury associated with blunt abdominal trauma, is best diagnosed by abdominal CT with intravenous contrast. As with splenic injury, most liver injuries can be managed without surgical intervention. However, when necessary, surgical control of bleeding is typically accomplished using conservative measures rather than resection.

Kidney injuries likewise rarely require surgical intervention in childhood, except in cases of uncontrollable hemorrhage, urine leak, or renal pedicle injury. Pancreatic injury occurs much less frequently than other solid organ injuries in children; it often is the result of impact with bicycle handlebars and may occur as an isolated injury. Abdominal CT is the diagnostic modality of choice. Management is typically nonoperative, unless the pancreatic duct is transected or major internal bleeding occurs.

Hollow organ injury occurs less frequently than solid organ injury and is more difficult to detect, so a high index of suspicion is required. Motor vehicle collisions in which children are restrained with poorly fitting lap belts may result in intestinal injury. Pneumoperitoneum or extravasation of oral contrast may be detected on abdominal CT. Often, free abdominal fluid is the only abnormality and remains a nonspecific finding. Similarly, motor vehicle collisions in which children are poorly restrained can also result in injury to the bladder. Disruption of the dome of the bladder, manifested by intraperitoneal extravasation of IV contrast, is repaired via laparotomy. Disruption of the remainder of the bladder, detected by extraperitoneal leakage of contrast, is treated via urinary or suprapubic catheter decompression.

When physical examination or abdominal CT findings are equivocal, a high index of suspicion should be maintained and follow-up serial examinations should be performed. Diagnostic laparoscopy may be helpful in selected cases.

Abdominal compartment syndrome, defined by a sustained intra-abdominal pressure of ≥20 mm Hg accompanied by new organ dysfunction, frequently manifests as abdominal distention and oliguria or anuria, respiratory decompensation, hypotension or shock, and metabolic acidosis. The syndrome is uncommon following major trauma in childhood and generally follows overzealous fluid resuscitation. As in adults, timely surgical decompression is required for optimal recovery.

E. Pelvic and Extremity Trauma

Case Study

A 5-year-old girl is an unrestrained passenger in a motor vehicle crash. Extrication from the damaged vehicle was prolonged. Vital signs were unobtainable at the scene, where she was immobilized with a semirigid collar and long spine board before transport. In the emergency department, the patient is severely agitated, moaning, and crying. Vital signs reveal a temperature of 96.6°F (36°C), a heart rate of 140 beats/min, a respiratory rate of 40 breaths/min, and a blood pressure of 70/45 mm Hg. Spo_2 is

94% on room air. Pelvic instability is detected on physical examination. The chest radiograph and head CT are normal, but the pelvic radiograph confirms multiple pelvic ring fractures. She additionally has a fractured left humerus.

Detection

– What maneuvers should be undertaken for control of bleeding?

– What additional injuries are likely to be present?

Intervention

– What are the most immediate treatment strategies?

– Should blood products be given?

– Should a Foley catheter be placed?

Reassessment

– Is the current treatment strategy effective?

– Does the patient require other therapeutic interventions for hemodynamic stability?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How should this treatment strategy be implemented?

– Who is to do what and when?

Musculoskeletal trauma rarely results in life-threatening hemorrhage in children. Exceptions to this rule include unstable pelvic fractures and bilateral femur fractures. Application of a pelvic binder or sling to limit pelvic volume is the first step in controlling retroperitoneal bleeding associated with pelvic disruption, followed by aggressive volume resuscitation with blood products. Ultimately, selective embolization by interventional radiology or external fixation may be required. Long bone fractures are immobilized as soon as possible to limit pain, lessen hemorrhage, and restore perfusion in cases where limbs are threatened by vascular compromise. Impaired limb perfusion is often associated with fractures of the elbow and knee; in-line fracture reduction may relieve external impingement upon arterial flow. Ischemic and crush injuries, most often of the distal lower extremities, may result in compartment syndrome, requiring urgent fasciotomy and aggressive hydration to preclude myoglobin deposition in the renal tubules and subsequent development of acute renal failure. Hyperkalemia may also result from major crush injuries and should be treated with administration of calcium and sodium bicarbonate.

Fractures of the pelvis and femur that occur when pedestrians are struck by moving vehicles may be associated with head and truncal injuries in a frequently incomplete pattern known as the *Waddell triad*. As previously stated, small children sustaining large force blunt trauma, such as in motor vehicle collisions, rarely sustain isolated injuries, so a careful search of the body core is warranted.

F. Soft Tissue Trauma

All open wounds require debridement of nonviable tissue, followed by primary closure if feasible or appropriate. If this is not possible, dressing changes and local wound care should be implemented, with delayed primary closure performed once granulation tissue has formed, usually within 5 to 7 days. Vacuum-assisted closure of large, open, contaminated wounds, using negative pressure wound therapy (eg, with reticulated open cell foam) may facilitate healing. Fasciotomy wounds can eventually be closed using a "shoelace" technique, though skin grafting may be needed if primary closure cannot be achieved. Wounds should be irrigated with large volumes of sterile normal saline (or sterile water) with a bulb device, gravity irrigation, or pulse lavage. Antimicrobial prophylaxis is warranted for all but clean, incisive wounds. Tetanus prophylaxis is warranted for all tetanus-prone wounds. All penetrating injuries must be considered contaminated and managed accordingly.

G. Drowning

The drowning process refers to the pathological effects that occur with submersion injury. Some still characterize drowning as "dry" or "wet," although these terms are less commonly used. Dry drowning occurs more frequently in adults. No fluid is aspirated into the lungs. Victims develop severe laryngospasm and often have significant mucus and froth in the upper airway. Death results from cerebral anoxia. Wet drowning involves the aspiration of fluid into the lungs with resultant pathophysiologic changes.

Following submersion, a period of voluntary apnea occurs. In young children, the initial contact with the water is accompanied by the diving reflex, which shunts blood flow from the skin and splanchnic circulations to the coronary and cerebral circulations. Blood pressure is increased and heart rate is decreased. Fear and the cold water temperature amplify the diving reflex, which may partially account for the improved survival seen following long submersion in cold water. In warm water (59°F-68°F [15°C-20°C]), the submersion time to fatality in humans is 3 to 10 minutes. In cold water (32°F-59°F [0°C-15°C]), neurologically intact survival has been reported in submersion as long as several hours. After the initial period of breath holding, a break point is reached and the victim will take a breath, resulting in aspiration of water. Initial water entry into the upper airway will lead to laryngospasm, which persists until hypoxia leads to muscle relaxation. At the same time, secondary apnea occurs, leading to involuntary gasping breaths, respiratory arrest, and eventually cardiac arrest. Given the possibility of good outcomes with cold water submersion injury, resuscitation should continue until the core temperature reaches 89°F to 95°F (32°C-35°C). The pulses may be very difficult to palpate in the setting of hypothermia and bradyarrhythmias.

Patients in the drowning process are managed much like other trauma patients. Severe electrolyte abnormalities are uncommon, and antibiotics are generally not indicated. Patients should be evaluated

for cervical spine injury only if the mechanism of injury is suggestive (eg, a dive into shallow water). The use of glucocorticoids is not indicated in the management of nonfatal drowning

Key Points

Traumatic Injuries in Children

- The primary patient survey consists of an ABCDE evaluation: airway and cervical spine immobilization, breathing, circulation and hemorrhage control, disability (neurologic) status and glucose level, and exposure/temperature management.
- Major hemorrhage should be aggressively treated with direct pressure/tourniquets, appropriate blood products, and hemostatic adjuncts until surgical hemostasis is obtained.
- Adjuncts to the primary survey include the E-FAST exam, cardiopulmonary monitoring, pulse oximetry, blood pressure monitoring, end-tidal carbon dioxide monitoring, blood gas analysis, urinary catheter placement, gastric catheter placement, and radiographs of the chest and pelvis.
- The secondary survey includes the medical (SAMPLE) history: symptoms, allergies, medications, past medical history and pregnancy status (as appropriate), last meal, and events and environment (mechanism of injury). The physical examination proceeds from head to toe.
- Adjuncts to the secondary survey include CT scans, completion of the cervical spine radiographs, radiographs of the thoracolumbar spine and extremities, ultrasonograms, and angiograms as indicated.
- Pulmonary contusions are common in children, but rib fractures are rare because of chest wall compliance. Because rib fractures are usually the result of significant trauma, detection in the absence of other significant trauma should raise a suspicion of abuse.
- The incidence of abdominal organ damage is higher in children than in adults. Splenic injury is most common, followed by liver injury. Intestinal injury is less common, and its diagnosis requires a high index of suspicion.

Suggested Readings

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PEDIATRIC BURN INJURY

Objectives

- Explain the pathophysiology of burn injury.
- Appropriately assess and initiate management of burn injuries in children.
- Recognize inhalation injury in children and initiate appropriate airway management.
- Recognize and appropriately manage the sequelae of burn injury.

Case Study

A 15-month-old girl is injured in a boiler explosion and partial building collapse. During initial assessment at a local emergency department, she is awake and crying, and her vital signs are remarkable for tachycardia and tachypnea. Physical exam demonstrates deep partial thickness burns involving 20% of her body surface area (BSA) and full-thickness burns involving 40% of her BSA. Approximately 40 minutes after arriving in the emergency department, the child produces black sputum and demonstrates stridor, respiratory distress, and somnolence. An arterial blood gas analysis reveals a normal Pao_2 of 99 mm Hg (13.2 kPa) and a $Paco_2$ of 100 mm Hg (13.3 kPa).

Detection

– What are the priorities of assessment?

– What types of injury are likely? Are external burns the only possible injury?

Intervention

– What treatment strategies should be initiated immediately?

Reassessment

– Is the current treatment strategy effective?

– How much fluid is required for resuscitation? What will indicate the adequacy of resuscitation?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– What consultations are necessary?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

I. INTRODUCTION

A burn is a traumatic injury to the skin or the upper aerodigestive tract that results from thermal energy or from chemical or physical agents. Deeper tissues, including muscles, nerves, and blood vessels, may also be injured if thermal energy results from an electrical source. Annually in the United States, approximately 1.2 million people sustain burns, and one-third of burn unit admissions and deaths occur in children. Children younger than 4 years are the subset of the pediatric population at the greatest risk of burn injury, and this population also suffers the greatest risk of abuse. Overall, scald injuries represent 65% of burns in children, while contact burns represent 20% of pediatric burn injuries. Despite improvements in burn care over the past 20 years, burn injury is still the fifth leading cause of unintentional injury-related death in children. Mortality is closely related to the depth and extent of injury and to age. Nearly half (47%) of all pediatric burn deaths are now due to sepsis, with respiratory failure (29%), anoxic brain injury (16%), and shock (8%) accounting for the remainder.

II. PATHOPHYSIOLOGY OF BURNS

The impact of a burn injury extends far beyond the involved areas of skin. The physiologic response to a burn can range from local inflammation in cases of limited injury to the systemic inflammatory response syndrome (SIRS) and, with more extensive injury, burn shock. Fluid loss from burned tissue is 5 to 10 times greater than that from healthy tissue and is accompanied by protein and electrolyte losses as well. Local edema is generated within 12 to 24 hours of injury and classically peaks between 24 and 48 hours. Some edema is always expected, but excessive fluid administration can contribute to pathologic edema if circumferential extremity burns are present. SIRS typically develops with burns of 15% to 20% BSA or greater. Temperature dysregulation, immune dysfunction with an attendant increased risk of wound infection and sepsis, and hypermetabolism are among the short- and long-term sequelae of significant burns. The progression of SIRS into multiorgan dysfunction/failure is the cause of high mortality rates in severe pediatric burn injury.

A. Approach

The approach to any emergency begins with an assessment of the airway, breathing, and circulation.

The assessment of the airway and a critical appraisal of the risk of smoke inhalation and airway injury are particularly relevant to victims of a closed space fire, who may sustain physical injuries at twice the rate of burn injuries, especially when caused by an explosive blast. Supplemental humidified oxygen should be administered empirically until a thorough and definite evaluation is completed. Once the airway is assessed and stabilized, IV access should be established quickly, preferably with 2 large-bore catheters. The volume of fluid resuscitation required is dictated by the depth and extent of injury.

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The progress of SIRS into multiorgan dysfunction/failure is the cause of high mortality in severe pediatric burn injury.

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B. Initial Management

1. Airway Assessment

Signs of inhalation injury include respiratory distress, hypoxia, stridor, wheezing, singed nasal hairs and eyebrows, drooling, oropharyngeal blisters, tongue swelling, and production of carbonaceous sputum. If these signs are present, early intubation should be performed to protect the airway before progression to airway obstruction. Inhalation injury results from inflammation and edema generated in response to the inhalation of toxic and/or superheated products of combustion. The inflammatory reaction can lead to the formation of endobronchial casts and obstruction of the distal airways. Mucociliary clearance mechanisms are disrupted, and the accumulation of necrotic debris and products of inflammation provide an environment conducive to infection. These patients often benefit from bronchoscopy to enhance pulmonary toilet and minimize the risk of pneumonia or, when pneumonia is present, facilitate its resolution.

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Signs of Inhalation Injury

- *Respiratory distress*
- *Hypoxia*
- *Stridor*
- *Wheezing*
- *Singed nasal hairs and eyebrows*
- *Drooling*
- *Oropharynx blisters*
- *Tongue swelling*
- *Production of carbonaceous sputum*

Inhalation injury may or may not be associated with carbon monoxide (CO) and cyanide (CN) poisoning. Any person exposed to fire in a confined space is at risk for these poisonings. CO poisoning may be responsible for as much as 80% of the deaths associated with inhalation injury. CO has a 250-fold higher affinity for hemoglobin than oxygen. It diminishes the oxygen-carrying capacity of hemoglobin and shifts the oxyhemoglobin dissociation curve to the left. It may also interfere with cellular oxygen metabolism at the mitochondrial level. Although measured partial pressures of oxygen are often normal in CO poisoning, there is relative tissue hypoxia. This is responsible for the delayed central nervous system sequelae of CO poisoning. This poisoning is diagnosed by measuring carboxyhemoglobin levels. Levels in excess of 5% are abnormal, and those greater than 25%, particularly when associated with headache or alteration in mental status, warrant referral to an institution where hyperbaric oxygen therapy can be delivered when feasible. In all cases, supplemental oxygen with a fraction of inspired oxygen of 1.0 should be administered. The half-life of carboxyhemoglobin is 3 to 4 hours in room air, diminishes to 30 to 60 minutes at a fraction of inspired oxygen of 1.0, and further decreases to 15 to 20 minutes under hyperbaric conditions at 3 atmospheres.

A high index of suspicion is necessary to diagnose CO poisoning because patients may present with nonspecific signs such as nausea, vomiting, headache, or altered sensorium. Any patient who suffers loss of consciousness at the scene of a fire should be assumed to have CO poisoning. All patients at risk should receive 100% oxygen. Carboxyhemoglobin levels do not reflect the tissue burden of CO, so patients with significant neurologic abnormalities should be referred for hyperbaric oxygen therapy whenever possible.

- *Diagnostic bronchoscopy is indicated in suspected inhalation injury to aid in definitive diagnosis.*
- *Therapeutic bronchoscopy facilitates removal of carbonaceous debris and the products of inflammation generated in response to inhalation injury.*

CN poisoning results from an acute energy deficit caused by the inhibition of mitochondrial cytochrome C oxidase, rendering the cell unable to utilize oxygen. Inhalation of significant amounts of CN may therefore result in coma, seizures, apnea, and death. The historic CN antidote kit consists of inhaled amyl nitrite and IV sodium nitrite to convert hemoglobin-bound iron from the ferrous to the ferric state, thereby creating methemoglobin, to which CN preferentially binds, converting methemoglobin to cyanmethemoglobin. This is then followed by IV sodium thiosulfate, which converts cyanmethemoglobin into thiocyanate, sulfite, and hemoglobin. However, hydroxocobalamin, the hydroxyl form of vitamin B_{12}, combines with CN to form the nontoxic cyanocobalamin form of

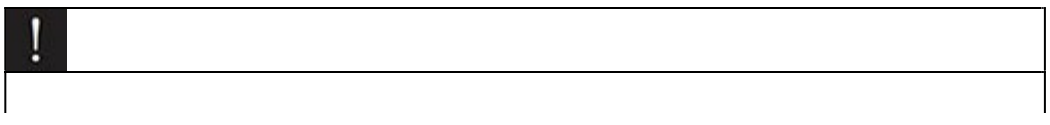
vitamin B_{12}, which is harmlessly excreted in urine. The advantage of hydroxocobalamin over the nitrites and sodium thiosulfate is that the former acts intracellularly and intravascularly, whereas the latter acts only intravascularly. Experience with hydroxocobalamin use in children remains limited, but continues to grow and is becoming the standard of care. As with the historic cyanide antidote kit, it must be administered as soon as possible following CN exposure for maximum benefit to be realized.

2. Fluid Resuscitation

The burn victim requires fluid resuscitation because the injury itself causes the elaboration of inflammatory mediators that produce local and systemic capillary leakage and intravascular volume loss. Superficial burns involving 10% to 15% of BSA can be managed with oral rehydration alone or with IV fluids for supplementation. Burns in excess of 15% BSA typically require IV fluid resuscitation, preferably with 2 large-bore IV lines. A bladder catheter should be inserted to facilitate accurate monitoring of urine output and to assess the adequacy of the resuscitation. IV catheters can be safely placed through burned tissue, but placement distal to circumferential burns should be avoided.

The volume of fluid resuscitation that is required is dictated by the depth and extent of injury and historically was most commonly guided by use of the Parkland formula, which estimates the patient's fluid deficit to be 4 mL/kg/% BSA burn in addition to the maintenance fluid requirement. First half of the calculated fluid deficit is given in the first eight hours. Second half is given in the next 16 hours. Alternatives to this formula which are increasingly being employed are the modified Brooke system, which recommends 2 mL/kg/% BSA burn over 24 hours for adolescents ≥14 years of age, 3 mL/kg/%BSA for children <14 years of age, and the Brooke system as modified by O'Neill, which recommends 2 or 3 mL/kg/% BSA burn plus maintenance IV fluids for infants and small children. Lactated Ringer solution or Hartmann solution is the initial resuscitation fluid of choice. Ultimately, fluid administration should be guided by repeated evaluation of the patient's intravascular volume, which is reflected in the heart rate, blood pressure, capillary refill, and urine output, and readjusted as frequently as indicated. Adequate urine output is 1.5 to 2 mL/kg/h for children younger than 2 years, 1 to 1.5 mL/kg/h for children between 2 and 10 years, and 0.5 to 1 mL/kg/h for children older than 10 years. Crystalloid is typically employed during the first 24 hours of resuscitation. The use of colloid in the initial resuscitation has not been shown to be beneficial. Albumin is often administered as a 5% solution at a daily dose of 0.3 to 0.4 mL/kg/% BSA, divided over 24 hours during the second 24 hours of burn resuscitation, in addition to maintenance IV fluids. As fluid resuscitation progresses, care must be taken not to overload the patient, as many burn victims receive a greater volume of fluid than that estimated by the given formulas.

Because electrical burns typically damage deep tissues below the body surface, particularly muscle tissue, rhabdomyolysis may result, which in severe cases may lead to acute kidney injury. In such situations, especially if myoglobinuria is observed, fluid resuscitation should commence at 4 mL/kg/% BSA to achieve an hourly urine output of 1-1.5 mL/kg/hr and minimize deposition of muscle pigments in renal tubules.



Fluid Resuscitation

First 24 hours

- *Parkland formula: 4 mL/kg/% BSA burn over 24 hours*
- *Modified Brooke system: 2 mL/kg/% BSA burn over 24 hours (≥14 years); 3 mL/kg/%BSA (<14 years)*
- *Modified Brooke system as further modified by O'Neill: 2 to 3 mL/kg/% BSA burn over 24 hours plus maintenance IV fluids for infants and small children*
- *Fluids ultimately titrated to:*
  – Heart rate
  – Capillary refill
  – Blood pressure
  – Urine output: 1.5 to 2 mL/kg/h (0-2 years), 1 to 1.5 mL/kg/h (2-10 years), 0.5 to 1 mL/kg/h (>10 years)

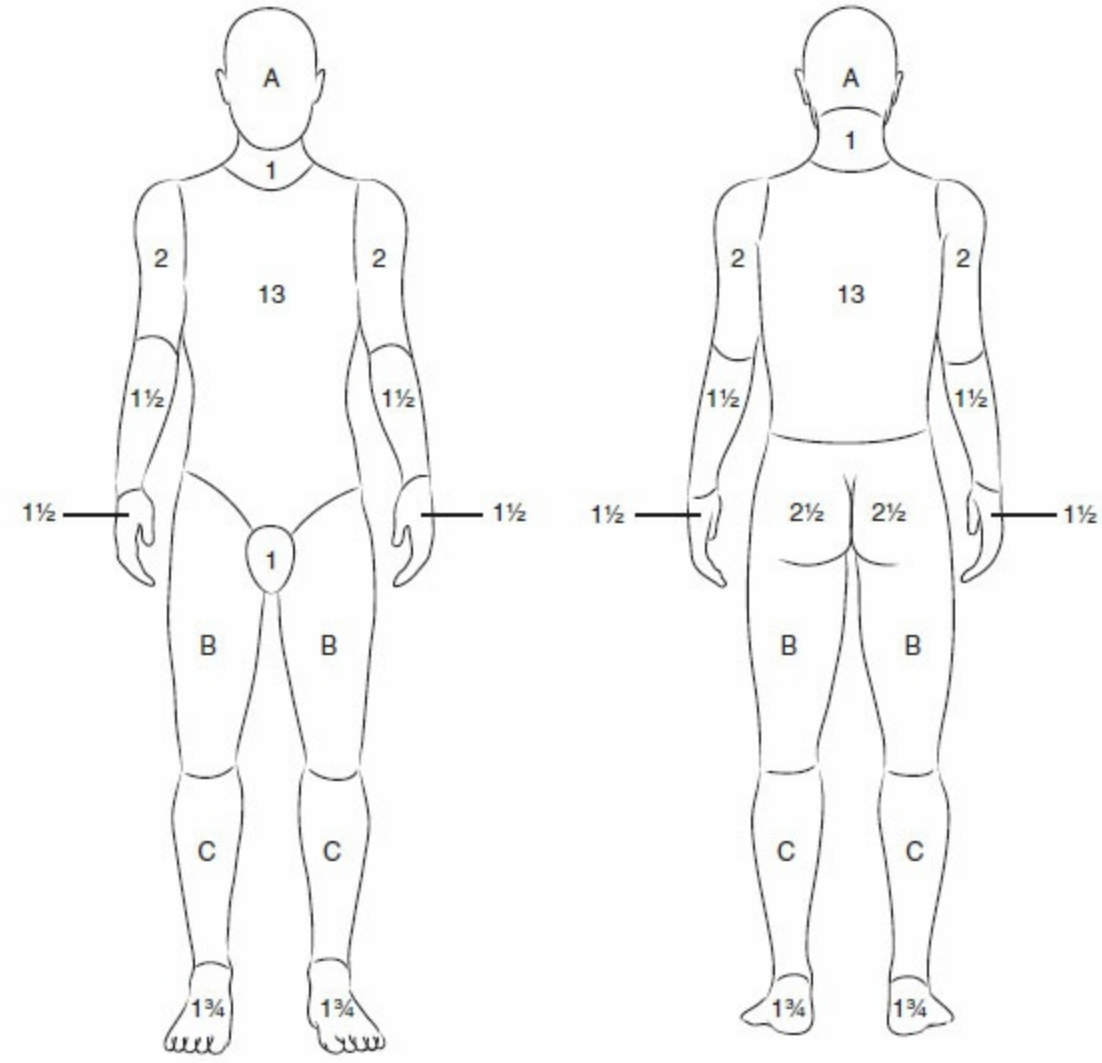
Second 24 hours

- *Maintenance IV fluids*
- *Still may need resuscitation fluid in addition to maintenance fluid, albeit less than in first 24 hours*

3. Assessment of Burn Injury

The most appropriate tool to estimate the involved BSA in children younger than 15 years is the commonly published and frequently used Lund-Browder chart (**Figure 10-1**). For young children with irregular splash burns, the rule of palms (which equates the entire palmar surface of the patient's hand, including the digits but excluding the wrist, to approximately 1% of BSA) may also be useful. The widely employed rule of nines is most appropriately applied to patients older than 15 years (**Figure 10-2**). Careful assessment of the depth and severity of injury is important because these factors heavily affect healing. The grading of burns as first, second, and third degree is imprecise and fails to accurately reflect the depth of injury. The current classification scheme, while more precise, is similar to the former insofar as it is also based on depth of injury relative to the anatomy of the skin. The categories are superficial, superficial partial thickness, deep partial thickness, full thickness, and deep full thickness (subdermal).

Figure 10-1. Lund-Browder Chart

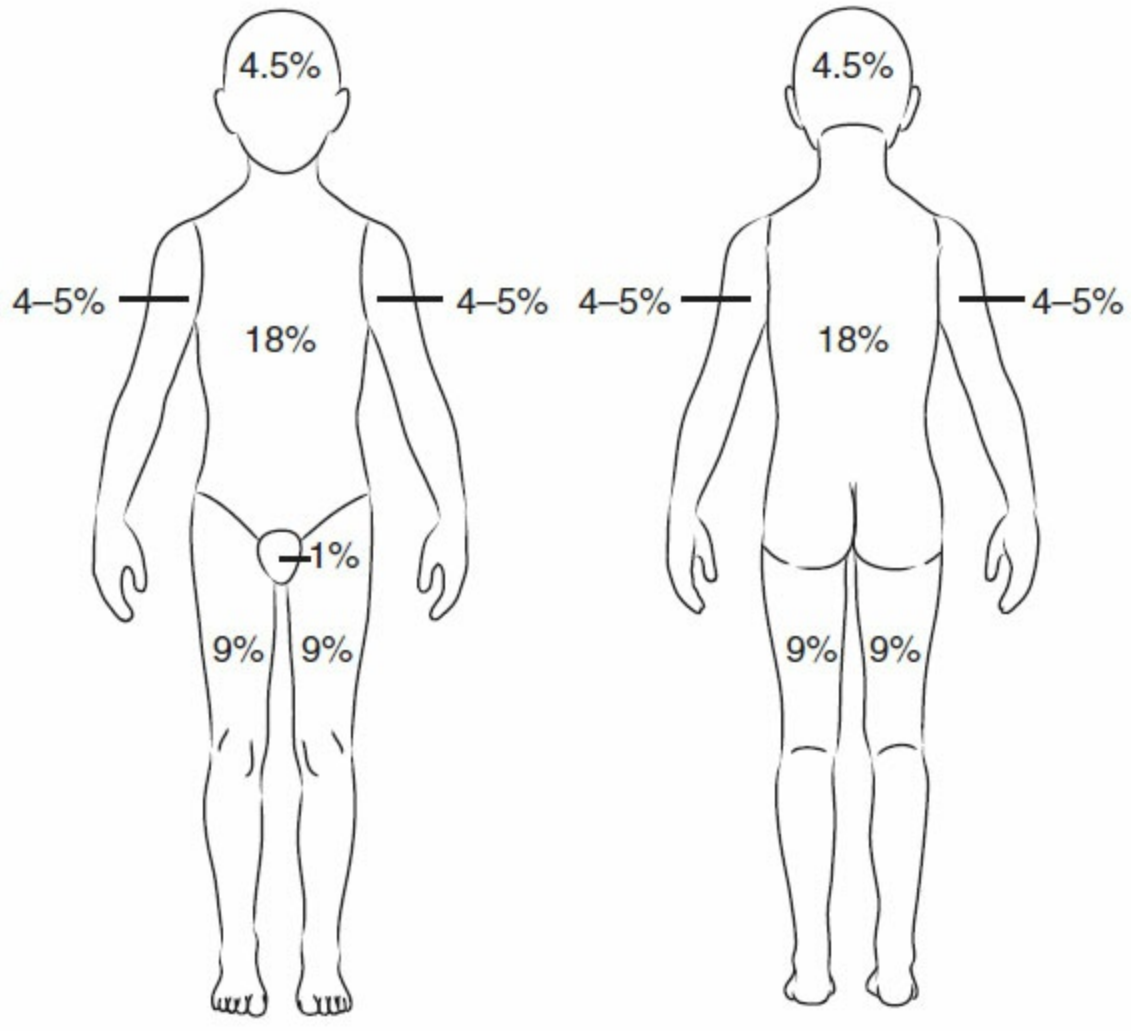


LUND-BROWDER CHART
Relative Percentage of Body Surface Area Affected by Growth

Age in years	0	1	5	10	15	Adult
A-head (back or front)	9½	8½	6½	5½	4½	3½
B-1 thigh (back or front)	2¾	3¼	4	4¼	4½	4¾
C-1 leg (back or front)	2½	2½	2¾	3	3¼	3½

The Lund-Browder chart is the most accurate method for estimating burn extent and must be used in the evaluation of pediatric patients under 15 years of age.

Figure 10-2. Rule of Nines



Most appropriate for children over 15 years of age.

The skin is composed of 3 layers: epidermis, dermis, and subcutaneous tissue. The epidermis is the outermost layer and consists of stratified squamous epithelial cells known as *keratinocytes*. The epidermis is avascular and receives its blood supply and nutrients from the dermis. The basement membrane lies between the epidermis and dermis. The dermis is the most physiologically active layer of skin. It consists principally of fibroblasts but also contains basal epidermal cells, macrophages, and neutrophils, and is structurally supported by collagen, glycosaminoglycans, and fibronectin. The dermis contains blood vessels, lymphatic channels, sebaceous and sweat glands, and hair follicles.

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Categorization of Burn Injury

Superficial partial thickness (formerly second degree)

- *Red, wet blisters*
- *Exquisitely painful*

Deep partial thickness (formerly second degree)

- *White or yellow appearance, dry*
- *Less pain and less blistering than superficial partial thickness*

Full thickness (formerly third degree)

- *White, yellow, black, or brown in color*
- *Firm and inelastic*
- *Distinctly not painful*

The local response to burn is characterized by zones of coagulation, stasis, and hyperemia (**Figures 10-3** to **10-5**). The zone of coagulation is the most central area of injury and exhibits the necrosis that results from greatest contact with the heat source. The zone of stasis, or injury, surrounds the zone of coagulation (necrosis). The microvasculature of this tissue is disrupted, making it prone to secondary ischemic injury. Tissue within the zone of stasis has the potential for regeneration, but its circulation must be supported with fluid administration for the first 24 to 48 hours to maximize the survival of viable tissue. The zone of hyperemia, which surrounds the zone of stasis, is the minimally affected outer border of injury. Such areas heal spontaneously in 7 to 10 days.

Figure 10-3. Superficial Partial-Thickness Burn

Superficial Dermal Burn
Characteristics
1. Necrosis confined to upper third of dermis
2. Zone of necrosis lifted off viable wound by edema
3. Small zone of injury
Zone of necrosis (coagulation)
Edema layer
Zone of injury (stasis)
Normal tissue

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Figure 10-4. Deep Partial-Thickness Burn

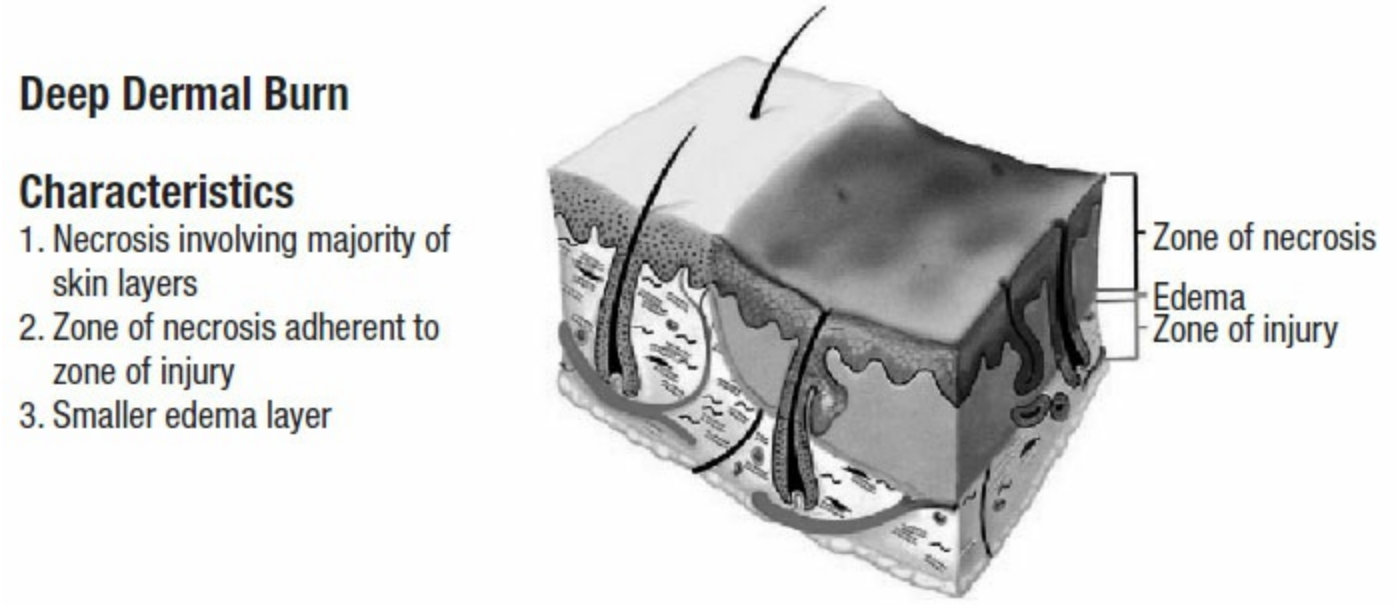

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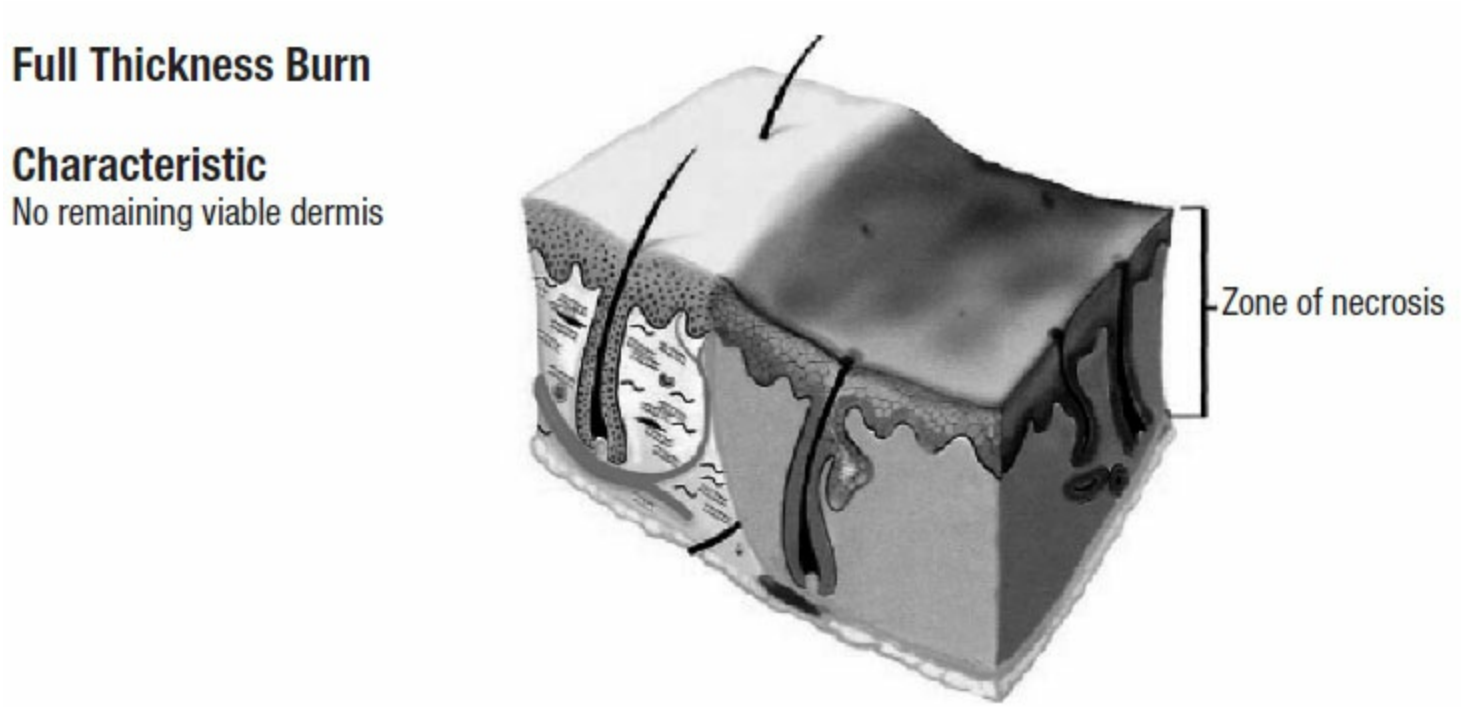
Figure 10-5. Full-Thickness Burn



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Methods for the assessment of the depth of burns are shown in **Figure 10-6**. It is important to recognize, however, that many burns will not be limited to a single depth level, but may involve one or more contiguous depth levels within a given burned area. For example, hot water scald and splash burns, the most common type of burns encountered in small children, are rarely homogeneous, and may encompass epidermal, superficial dermal, and even mid dermal elements, but much less often deep dermal elements. Only rarely will they encompass full thickness elements, unless the temperature and duration of contact are especially high or long. No published studies directly address the temperature and duration of hot water contact needed to cause scald burns in children, but the U.S. Consumer Product Safety Commission (CPSC) states that full thickness burns in adults will occur in 2

sec at 150°F, in 6 sec at 140°F, in 30 sec at 130°F, and in 5 min at 120°F (https://www.cpsc.gov/s3fs-public/5098.pdf). Given their thinner skin, the temperature and time needed to cause hot water scald and splash burns in small children is likely lower and shorter.

Figure 10-6. Methods of Assessment of the Depth of Burn

Depth	Colour	Blisters	Capillary Refill	Sensation	Healing
Epidermal	Red	No	Present	Present	Yes
Superficial Dermal	Pale Pink	Present	Present	Painful	Yes
Mid Dermal	Dark Pink	Present	Sluggish	+/-	Usually
Deep Dermal	Blotchy Red	+/-	Absent	Absent	No
Full Thickness	White	No	Absent	Absent	No

Australian and New Zealand Burns Association. *Emergency Management of Severe Burns: Course Manual*. 18th ed. Albany Creek, Queensland, Australia: Australia and New Zealand Burns Association; 2016.

Epidermal (superficial) burns, formerly called *first-degree burns*, involve only the epidermis. They are typically dry and characterized by erythema and pain without bullae. The keratinocytes slough, and epithelial cells migrate to the surface to facilitate wound closure. These injuries typically heal spontaneously within 5 to 7 days. They are managed with topical analgesics and moisturizing creams. They are not customarily included in estimating the extent of BSA burned because they heal completely without intervention.

Superficial, mid, and deep dermal (partial thickness) burns, formerly called *second-degree burns*, extend through the epidermis and into the dermis. Because dermal burns are not homogeneous and can evolve over hours to days, debridement is usually necessary to accurately assess the depth of injury. Superficial dermal and mid-dermal burns, are erythematous and moist, and the presence of blisters is a hallmark. They are exquisitely painful because of the exposed and injured nerves. They heal via re-epithelialization from the uninjured deeper elements of hair follicles usually within 2 to 3 weeks.

Deep partial-thickness burns, characterized by a white or yellow appearance, may be less painful and demonstrate less blistering than superficial partial-thickness burns. As the depth of injury increases, the amount of pain decreases because more nerve endings are destroyed. Without intervention, deep partial-thickness burns can heal by epithelialization and contraction in 3 to 6 weeks. However, because the results are functionally and cosmetically poor, deep partial-thickness burns are typically managed with tangential excision and grafting.

Full-thickness burns, formerly called *third-degree burns*, usually result from flame, prolonged contact, or hot oil or grease. Scalding, without prolonged exposure to hot water, rarely produces such

burns. Full-thickness burns are characterized by white, yellow, brown, or black discoloration. Severe edema without blisters is seen. The texture of the skin is firm and inelastic, and eschar may be present. Deep full-thickness, or subdermal, burns extend to the fascia and muscle with the potential to involve tendon and bone. Usually seen in victims of house fires, deep full-thickness burns require skin grafting and may need musculocutaneous flaps. In cases of circumferential full-thickness burns of the chest or extremities, early escharotomy may be needed to permit adequate ventilation and perfusion, respectively. Of note, a special type of full-thickness burns are those caused by electrical current. Fasciotomy may also be required in such cases to relieve pressure on swollen muscle tissue, in addition to the high-volume fluid resuscitation described above, and early transfer to a burn center.

III. TREATMENT OF BURNS

A. Criteria for Hospital Admission

The indications for a patient to be admitted to the hospital for burn care include superficial partial-thickness burns in excess of 10% BSA or to critical areas such as the eyes, face, ears, hands, feet, or genitals, an inability to maintain adequate hydration or nutrition, caregivers who are unwilling or unable to provide burn care, burn injury in a child with a significant chronic medical condition, the need for IV analgesics or anxiolytics, and cases of suspected child abuse. The American Burn Association has established and published criteria for referring patients to burn centers (**Table 10-1**).

Table 10-1	AMERICAN BURN ASSOCIATION INDICATIONS FOR REFERRAL TO A BURN CENTER

- Partial thickness burns >10% total body surface area
- Burns that involve the face, hands, feet, genitalia, perineum, or major joints
- Third-degree burns in any age group
- Electrical burns, including lightning injury
- Chemical burns
- Inhalation injury
- Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality
- Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
- Burned children in hospitals without qualified personnel or equipment for the care of children
- Burn injury in patients who will require special social, emotional, or rehabilitative intervention

Classified using: American Burn Association/American College of Surgeons. Guidelines for the operation of burn centers. *J Burn Care Res*. 2007;28:134-141.

B. Nonsurgical Wound Care

Among the goals of wound care are minimizing bacterial colonization and preventing infection. A variety of topical agents may be applied to superficial partial-thickness burns and deep partial-

thickness or full-thickness burns before skin grafting to prevent wound infection (**Table 10-2**). These agents include bacitracin, silver sulfadiazine, mafenide acetate, and silver-impregnated antimicrobial dressings. The choice of topical agent or dressing has evolved over time based on research findings. Agents should be chosen in consultation with the burn center staff, if the patient is referred, as some agents will obscure the burn and may need to be removed for evaluation.

Table 10-2 TOPICAL AGENTS EMPLOYED IN BURN CARE

Product	Application	Advantages	Disadvantages
Bacitracin	Superficial or deep partial-thickness burns, small areas	Not water soluble, good on face	Not indicated for large surface area
Creams			
Silver sulfadiazine	Deep partial- or full-thickness burns	Soothing, good for range of motion	Possible neutropenia, little eschar penetration
Mafenide acetate	Deep partial- or full-thickness burns	Penetrates eschar	Painful, metabolic acidosis
Solutions			
Aqueous silver nitrate (0.5%)	Deep partial- or full-thickness burns	Effective antimicrobial agent	Hyponatremia, stains wound, little penetration
Mafenide acetate (5%)	Graft dressing and open wound soak	Broad activity, moist dressing	
Impregnated Dressings			
Silver-containing antimicrobial barrier layer dressing (e.g., Acticoat)	Deep partial-thickness burn	Dressing change every 3 days (Acticoat 7 can be left on for 7 days)	Only deep partial-thickness burn
Silver-containing antimicrobial barrier dressing with absorptive wick-like properties (e.g., Aquacel Ag)	Deep partial-thickness burn	May be left on for 21 days	Less flexibility and ease of motion, only deep partial-thickness burns

Bacitracin effectively protects against Gram-positive and a few Gram-negative organisms but may produce local irritation. Silver sulfadiazine is the most commonly used topical antimicrobial in burn wound management. It is bactericidal against Gram-positive and Gram-negative organisms and yeast. It is indicated for partial-thickness and full-thickness burns in anticipation of grafting. However, silver sulfadiazine is to be avoided in patients with sulfa allergies and glucose-6-phosphate dehydrogenase deficiency. Because it causes permanent skin staining, it is contraindicated for facial burns in children younger than 2 months. It is also contraindicated in late-term pregnant women due to the association of kernicterus and sulfonamides. Mafenide acetate is a bacteriostatic agent effective against both Gram-positive and Gram-negative organisms, including *Pseudomonas* and certain anaerobes. It is employed primarily in partial-thickness or full-thickness burns because it can penetrate devitalized tissue. It is active in acidic (purulent) environments and is therefore particularly appropriate for eschar. Mafenide acetate can produce metabolic acidosis because it inhibits carbonic anhydrase.

A variety of relatively new, silver-impregnated, antimicrobial dressings with the ability to absorb exudate may be applied to burn wounds. The advantage of these dressings over the topical

antimicrobials is that they can be left in place for several days, which eliminates the need for daily dressing changes and may in turn be associated with less pain and anxiety, thereby decreasing the need for sedation and analgesia. As yet there are no good comparative outcome data, but antimicrobial dressings appear to be equal in efficacy and cosmetic outcome compared to topical antimicrobial agents.

C. Burn Wound Excision and Grafting

Although no controlled studies demonstrate improved outcomes with early tangential excision and grafting of burn injuries, this practice is likely responsible for the decreased mortality from burn injuries realized over the past 20 years. The rationale for early closure of deep injuries is to reduce local infection and fluid loss and their attendant complications.

Current practice calls for tangential excision and grafting within 1 to 2 weeks of the injury. This should be performed in an operating room under general anesthesia by appropriately qualified surgeons, preferentially at a burn center. Tangential excision is often associated with significant blood loss, which should be anticipated. For large burn wounds, therefore, tangential excision and grafting may need to be done in stages, balancing the risks of significant blood loss against those of repeated exposure to general anesthetics, especially in children younger than 3 year of age.

For the young child with deep or large partial-thickness injury, it is often necessary to provide moderate to deep sedation, in addition to analgesia, to accomplish appropriate debridement and dressing changes. The depth of sedation necessary will depend on the extent of the injury, debridement required, the patient's age, and respiratory and cardiovascular status.

D. Management of Inhalation Injury

Therapy for inhalation injury consists of aggressive pulmonary toilet, use of mucolytics, early identification and treatment of infection, and supportive care. Although no data support the routine use of mucolytics in patients with inhalation injury, many experts recommend *N*-acetyl-cysteine (3-5 mL of a 20% solution nebulized 3-4 times a day) or dornase alfa (2.5 mg nebulized once or twice daily) to help clear tenacious secretions. Routine prophylaxis with antibiotics is not used; corticosteroids are of no benefit and are potentially harmful.

The level of respiratory support that is required may range from supplemental oxygen to advanced modes of assisted ventilation and hyperbaric oxygen therapy. Supplemental humidified oxygen should be provided via nasal cannula or simple face mask. If stridor is present due to upper airway inflammation and edema, racemic epinephrine may be employed to transiently relieve the obstruction to airflow. Helium-oxygen admixtures (heliox) reduce resistance to turbulent airflow and, in turn, decrease the work of breathing in situations of upper airway obstruction. When the airway is compromised, however, these patients should be intubated for airway control. Based on a recent survey of pediatric burn centers, approximately 12% of pediatric burn victims require intubation, with approximately 70% of those intubated having sustained inhalation injury.

Current pediatric advanced life support guidelines support the use of cuffed tubes in all children.

Ample evidence suggests that cuffed endotracheal tubes are safe and more effective in critically ill and burned pediatric patients, especially those with restrictive lung disease, in whom the presence of a leak around the endotracheal tube would impair oxygenation and ventilation. Tube size for cuffed tubes may be calculated using the formula: (age/4) + 3.5. However, children with burns and/or inhalation injury frequently experience significant airway edema; therefore, the size (internal diameter) of the endotracheal tube may need to be reduced by 0.5 mm.

The prevalence of acute respiratory distress syndrome in mechanically ventilated adults with major burns has been estimated to be as high as 54%. Although the prevalence in pediatric burn patients is likely lower, acute lung injury and acute respiratory distress syndrome represent significant clinical challenges, especially in very young patients. The level of pulmonary dysfunction should govern the mode of mechanical ventilation employed in the care of these patients. Lung-protective ventilator strategies should be used in those at risk for acute lung injury. Low tidal volume/high positive end-expiratory pressure is used initially and progresses to high-frequency oscillatory ventilation or airway pressure release ventilation in patients who require positive end-expiratory pressure in excess of 10 to 15 cm H_2O. Additional information on ventilator management may be found in the chapter on mechanical ventilation (**Chapter 5**).

Tracheostomy is classically performed in patients following a prolonged course of endotracheal intubation. Although early tracheostomy (2-4 days after initiation of assisted ventilation) has also been used safely in a cohort of pediatric burn patients, the procedure is generally reserved for patients who have failed extubation or for those projected to require chronic mechanical ventilation, such as neurologically devastated patients.

E. Hypermetabolism and Nutrition

The classic description of the metabolic response to injury includes an early hypometabolic "ebb," or catabolic phase, characterized by low cardiac output and a decreased metabolic rate, followed by a hypermetabolic "flow," or anabolic phase, that starts 24 to 36 hours after injury. Protein metabolism and energy expenditure increase by approximately 50% due to hypermetabolism, fluid losses, sepsis, and inflammation in children following a large burn injury, and this catabolic state can persist for 9 to 12 months. Early and aggressive nutritional therapy has been shown to reduce the elevated resting energy expenditure in burn victims. The most effective method of determining this expenditure is by indirect calorimetry; when that is not available, nutritional therapy is usually guided by a formula (**Table 10-3**).

Table 10-3	SAMPLE NUTRITIONAL THERAPY FORMULAS
Age	Calculation
Infants (0-12 months)	2100 kcal/m² +1000 kcal/m² burn
Children (1-11 years)	1800 kcal/m² + 1300 kcal/m² burn
Children (12 years and older)	1500 kcal/m² + 1500 kcal/m² burn

Reprinted from *Burns*, 8, Rose JK, Herndon DN, Advances in the treatment of burn patients, S19-S26, Copyright (1997), with permission from Elsevier.

The enteral route is preferred for administration of nutrition. A nasogastric or nasoduodenal tube should be placed as soon as the initial evaluation and burn resuscitation are complete. In the absence of contraindications, enteral feeding should be initiated within 24 hours of injury. Feeding intolerance, as evidenced by significant gastric residual volume and diarrhea, may limit use of the gastrointestinal tract for caloric delivery. Diarrhea is a commonly encountered problem in the burn population. The etiology is likely multifactorial, usually noninfectious, and not worsened by hypoalbuminemia. Factors associated with a decreased incidence of diarrhea in burn victims include fat intake less than 20% of overall caloric intake, vitamin A intake greater than 10,000 IU per day in adults, and implementation of enteral feeding within 48 hours of burn injury.

In addition to vitamin A, other vitamins, minerals, and trace elements may be required in excess of recommended daily allowances, including calcium, magnesium, vitamin D, zinc, copper, and other micronutrients. Supplementation of calcium and magnesium is recommended until serum levels are within the normal range.

In addition to early and aggressive nutritional support, attempts have been made to pharmacologically mitigate the hypermetabolic state that persists following burn injury. The hypermetabolism, which can be blunted to some extent by β-blockade, has led some to recommend the use of propranolol together with human growth hormone. However, these drugs should be used only in burn centers experienced in the care of children.

Attempts have also been made to accelerate healing through the use of anabolic agents, such as oxandrolone. A recent single-center prospective trial has shown that administration of this agent improves the long-term recovery of severely burned children in terms of height, bone mineral content, cardiac work, and muscle strength, likely through decreased resting energy expenditure and increased insulin-like growth factor, benefits which appear to persist for up to 5 years following the burn, without evidence of deleterious side effects. Again, use of this agent should be limited to burn centers experienced in the care of children.

F. Hypoalbuminemia

In burn patients, hypoalbuminemia is a frequent finding. The etiology is multifactorial. Increased losses of albumin occur directly via drainage from wounds and as a consequence of profound capillary leakage, ignited by the cascade of inflammatory mediators triggered by the burn. Albumin production is also reduced in critical illness, likely due to an increase in production of acute phase proteins. Additionally, in the immediate post-resuscitation phase, a dilutional contribution to hypoalbuminemia may occur if intravascular volume is increased. Chronic illness and malnutrition are other potential causes of non-acute hypoalbuminemia.

Albumin contributes 80% of the normal colloid oncotic pressure; therefore, hypoalbuminemia is associated with edema, particularly of the pulmonary interstitium and bowel wall. Albumin is often administered in an effort to avoid exacerbating acute lung injury, diarrhea, feeding intolerance, impaired wound healing, and the resultant complications. However, the evidence demonstrates that mild to moderate hypoalbuminemia is well tolerated in previously healthy patients. Among burn patients with hypoalbuminemia, serum albumin gradually normalizes, especially in the context of aggressive nutritional therapy, and higher serum albumin concentration is seen in patients who receive

enteral, as opposed to parenteral, nutrition. Therefore, routine albumin repletion may not be warranted. The current recommendation is to ensure appropriate caloric delivery, preferably by the enteral route, as quickly as possible. In critically ill pediatric patients, 25% albumin may be added if the serum level is below 2 mg/dL.

G. Glycemic Control

As noted previously, protein metabolism and energy expenditure increase by approximately 50% in children following a large burn injury. The etiology of this hypermetabolic state is multifactorial but favorably affected by insulin administration. Hyperglycemia in adult burn patients is associated with increased morbidity and mortality rates. Although it has not been demonstrated that intensive insulin therapy can be safely and effectively implemented in the pediatric population, its use in the pediatric burn population may lower infection rates and improve survival. Careful attention must be paid to avoiding hypoglycemia. Members of the healthcare team participating in the care of pediatric burn patients should be sensitized to the importance of glucose monitoring and the recognition of hypoglycemia, especially in very young or nonverbal children. Standing insulin orders should only be written for those who demonstrate persistent hyperglycemia while they receive constant glucose delivery in the form of enteral tube feedings or parenteral nutrition; any orders must be associated with frequent bedside monitoring of serum glucose.

H. Pain Management in Pediatric Burns

Most children are managed with intravenous morphine infusions and acetaminophen (15 mg/kg four times daily). Dressing changes can be especially painful, for which ketamine may be a useful alternative. Oral analgesia in the form of morphine sulfate controlled-release (MS Contin), tramadol (Ultram) and oxycodone can also be used, but should be avoided if possible. Dressing changes can be especially painful, for which ketamine may be a useful alternative. The implementation of a patient-controlled analgesia (PCA) device is helpful in older patients who can cooperate with the instruction of use. In general, non-opioid analgesic medications should be initiated and used in combination. Other adjuvant medications include methadone, clonidine, and ibuprofen. A complete review of these medications can be found in the chapter on sedation and analgesia (**Chapter 20**).

IV. OUTCOME

Mortality from burns has improved over the past 20 years but may have reached a plateau in the past decade. The risk of death in children remains inversely proportional to age and directly proportional to the extent of injury. In children older than 2 years who suffer burns to >50% BSA, mortality is approximately 20%; in younger children, mortality is approximately 50%. Early excision and closure of injured tissue is believed to be the reason for improved mortality. Smoke inhalation and thermal injury increase the risk of death by 20%.

Care of the pediatric burn victim has advanced in parallel with advances in burn and critical care. Early physical therapy, splinting of injured extremities, and advances in grafting techniques and skin

substitutes all contribute to improvements in the quality of life following burn injury. Additional advancements in the elasticity of grafted tissues and microvascular reconstructive techniques promise to yield further improvements in cosmetic and functional outcomes.

Key Points

Pediatric Burn Injury

- Initial evaluation of pediatric burn patients begins with assessment of airway, breathing, and circulation. In addition, the extent and depth of burn injury must be assessed.
- Assessments for inhalation injury, smoke inhalation, and CO and CN poisoning are indicated; treatment should be initiated if appropriate.
- SIRS, a common finding in burn victims, contributes to intravascular volume depletion and shock.
- Adequate fluid resuscitation, in the first 24 hours and beyond, is important to avoid early complications of serious burns.
- Fluid administration may be guided by the Parkland formula or modified Brooke formulas, but ultimately must be titrated to urine output and perfusion.
- Early excision and grafting are now standards of care in the management of deep partial-thickness and full-thickness burns.
- Hypermetabolism is a significant long-term (9-12 months) sequela of significant burn injury, but appropriate management can significantly improve morbidity and mortality rates.

Suggested Readings

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"ABUSIVE" TRAUMA: DIAGNOSIS AND MANAGEMENT

Objectives

- Identify risk factors for abuse.
- Recognize injury patterns suggestive of abuse.
- Outline the appropriate evaluation of the child with suspicious injuries.
- Describe the initial resuscitation of a child with potentially abusive injuries.
- Review the healthcare provider role as a potential/mandated reporter of suspected child abuse.

I. INTRODUCTION

Child maltreatment is a tragically common cause of death, permanent disability, and suffering worldwide. Although many children present with histories and clinical findings that suggest abusive injuries, diagnosis and management may be complicated by atypical and delayed presentations, vague and misleading histories, and the patients' inability to communicate. Maltreatment must be considered in the differential diagnosis of any child presenting with injuries or a decreased level of consciousness. The term *"nonaccidental" trauma* is often used to describe inflicted injuries associated with child abuse or maltreatment. Use of this term, however, implies that unintentional trauma is "accidental," although it is now well established that most injuries are not "accidents" in that the majority of them could have been prevented.

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Abusive injuries are more likely to occur in stressed households, particularly when parent-child bonding is impaired.

!

Recognized risk factors for child maltreatment are varied, as can be seen in **Table 11-1**. Premature birth may interfere with infant-parent bonding. Infants discharged from the nursery after a prolonged

hospitalization may have significant impairments, including developmental delays, feeding problems, and respiratory difficulties. They may be irritable and difficult to console, and may ultimately exhaust the ability of caregivers to cope. Toileting mishaps and irritating behaviors may result in abusive injuries to toddlers and other children in the household.

Table 11-1 RISK FACTORS FOR CHILD MALTREATMENT

- Intimate partner violence
- Prior child abuse/neglect
- Substance-abusing caregivers
- Colicky/fussy child
- Prematurity or prolonged neonatal hospitalization
- Neurodevelopmental disability

II. INJURIES THAT RAISE SUSPICION OF CHILD ABUSE

Caregivers may report mechanisms of injury that are highly unlikely at the child's developmental stage. Older, often preverbal siblings may be blamed for injuries. Caregivers with substance abuse problems, particularly those involved in the production or sale of methamphetamine or other illegal substances, may be reluctant to summon emergency medical services to the home and may opt to transport an injured child themselves despite the added risk to the child.

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Inconsistencies relating to the circumstances of an injury and any delay in seeking care should raise concerns of child maltreatment.

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A. Patterns Suggestive of Abuse

Although patterns of physical injury are not absolute, among the injuries consistently found in children with witnessed, unintentional injuries are epidural hematomas; simple, linear skull fractures; diaphyseal and spiral fractures in ambulatory children; and irregular, pattern-lacking, asymmetric thermal injuries that have splash marks and do not spare the popliteal or antecubital fossae. A comparison of some common unintentional and abusive injuries is presented in **Table 11-2**.

Table 11-2 UNINTENTIONAL VERSUS ABUSIVE PATTERNS OF INJURY[a]

Site of Injury	Unintentional Injury	Abusive Injury

Head	Focal injuries at site of impact Clear history of causative incident Simple, linear skull fracture Absence of other injuries	Global/widespread injuries Inconsistent description of injury Diastatic, depressed skull fractures from reported short falls Injuries to other body sites
Skeletal	Mid-shaft clavicular Diaphysis of long bones in ambulatory children	Posterior ribs Metaphyses of long bones in nonambulatory children Vertebral body Multiple sites Injuries of varying ages
Cutaneous	Bruises over bony prominences in ambulatory children Burns with splash appearance	Bruises to face or head in nonambulatory children Bruises over padded areas, ears, neck, genitalia Patterned injuries and bites Burns with well-demarcated edges Bruises or burns involving genitalia

[a] No pattern of injury is diagnostic of either unintentional or abusive injury without consideration of the supporting history and developmental stage of the child.

Although unintentional impact injuries to the head can result in subdural hematomas adjacent to the site of impact, such hematomas are uncommon in falls of less than 3 to 4 feet. Conversely, diffuse thin-rimmed subdural hematomas, retinal hemorrhages, posterior rib fractures, and metaphyseal fractures are more commonly found in children with abusive injuries. Burns that are bilateral, symmetric, or of uniform depth; that have clearly defined borders, definable patterns, or evidence of healing or infection; or that include the anogenital areas should raise suspicion of maltreatment (**Figures 11-1** and **11-2**).

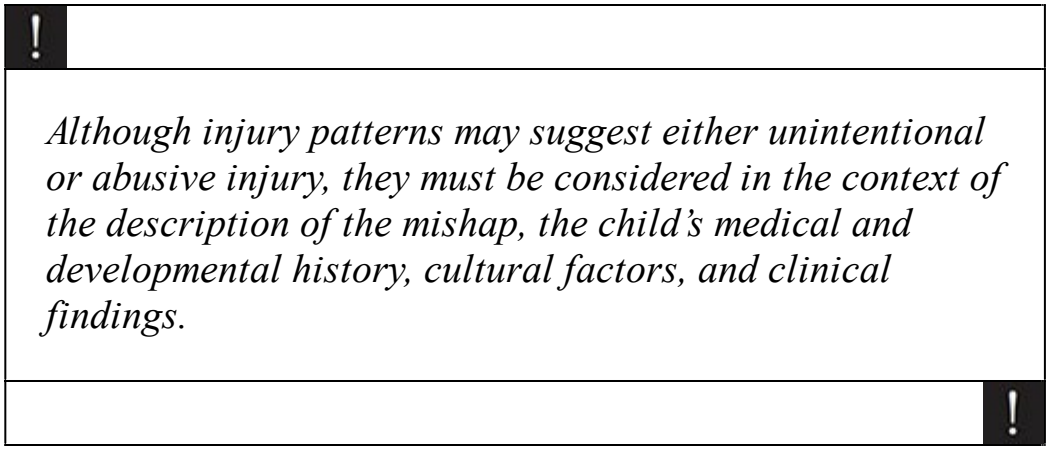

!
Although injury patterns may suggest either unintentional or abusive injury, they must be considered in the context of the description of the mishap, the child's medical and developmental history, cultural factors, and clinical findings.
!

Figure 11-1. Burns Caused by Immersion in Hot Water

Full-thickness burns with flexure sparing due to immersion in hot water.

Figure 11-2. Small, Healed, Patterned Burns

B. Common Patterns of Maltreatment

Case Study

A 2-month-old, previously healthy infant girl is brought to the hospital for evaluation of apnea and lethargy after she choked during a feeding. The mother states that the child started to have episodes of colic over the past week but was otherwise well before the mother went to work earlier that morning, leaving the baby and an older sibling in the care of her boyfriend. On initial examination, the infant is tachycardic with cool extremities, and respirations are irregular. The infant is immediately intubated and given fluid resuscitation. Although the secondary survey does not show any bruises, her anterior fontanelle is tense. Retinal hemorrhages are noted.

Detection

– What is the possible diagnosis?

Intervention

– What else needs to be done to resuscitate this infant?

– What diagnostic studies are indicated?

Reassessment

– What tests would be useful to evaluate the severity of the patient's condition?

– Have peripheral perfusion signs improved with resuscitation?

– Does the infant require other pharmacologic or surgical therapeutic interventions?

Effective Communication

– Where is the best place to manage this patient?

– Are other subspecialty consultations needed?

Teamwork

– How are you going to implement the treatment strategy?

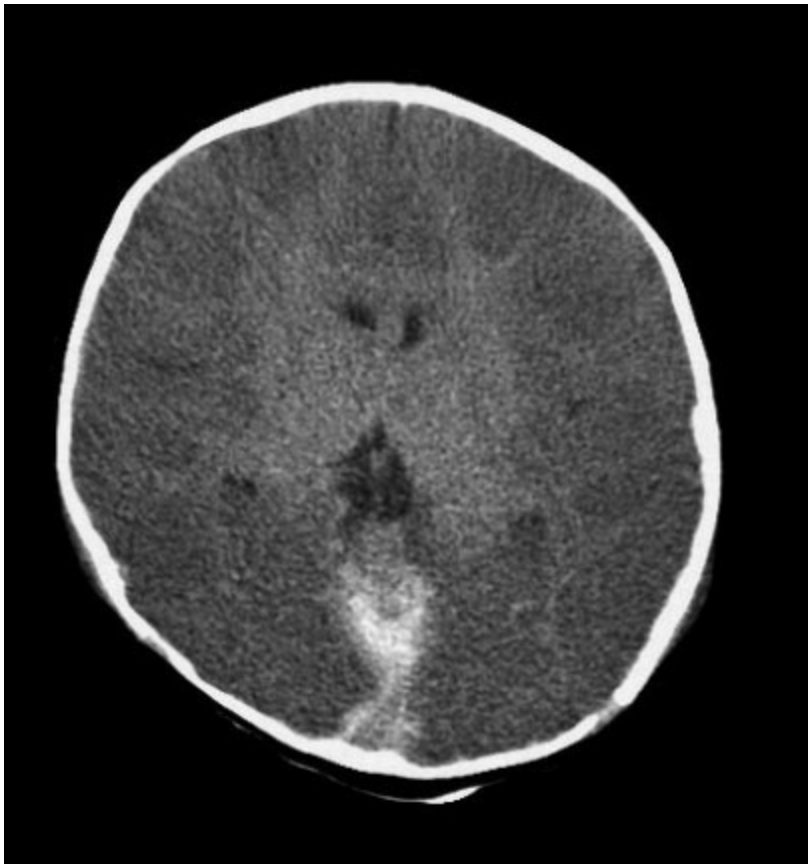
– Who is to do what and when?

1. Abusive Head Trauma

Because abusive head trauma (AHT) has been identified by varying terminology, information on this diagnosis is inconsistent and the actual number of children reported affected is unknown; this has limited the ability of the public health community to respond to the problems. In 2012, the Centers for Disease Control and Prevention came out with a definition for uniformity: AHT is an injury to the skull or intracranial contents of an infant or young child (<5 years) due to an inflicted blunt impact and or violent shaking. The most recent shift in terminology also reflects an attempt by the American Academy of Pediatrics to encourage physicians to utilize more general terms in describing the array of abusive mechanisms that can result in a pediatric head injury.

AHT is a frequently incomplete triad consisting of inflicted head injury with extra-axial hematoma, retinal hemorrhage, and skeletal injury, including metaphyseal and posterior rib fractures (**Figure 11-3**). Young infants are at the highest risk, but toddlers may display this pattern of abuse as well. A frustrated caregiver's violent shaking of a child, with or without concomitant projection onto a hard or soft surface, results in severe rotational and shearing forces capable of tearing bridging veins in the subdural and subarachnoid spaces. Recent evidence suggests that, in certain cases, shearing injury to the proximal cervical nerve roots may also be a contributory factor. Subdural bleeding is the most common intracranial finding in AHT victims (77%-90%); however, other intracranial pathologies can be seen in AHT.

Figure 11-3. Radiograph From an Infant With Abusive Head Trauma



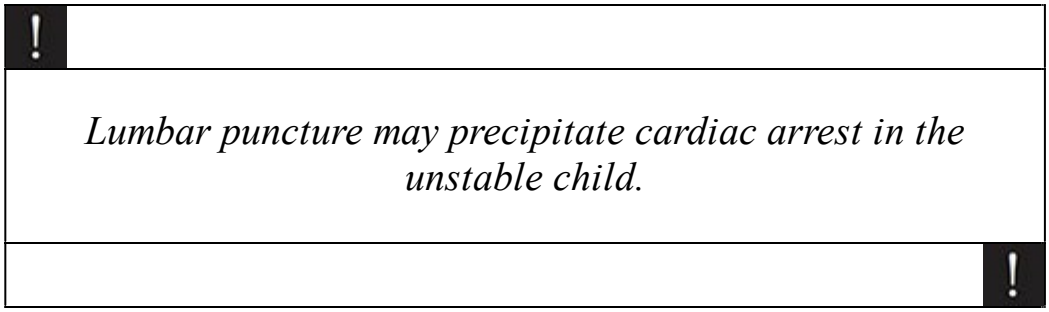
A radiograph reveals subarachnoid and subdural hemorrhages and cerebral edema consistent with abusive head trauma.

Unlike children with unintentional head injuries, who often present with clear histories and focal injury patterns, those with abusive head injuries may present with more global cerebral findings, clinical features consistent with shock, and pallor. Caregivers commonly deny or minimize trauma. Soft-tissue swelling and bruising may or may not accompany severe abusive head injuries, and bulging, tense fontanelles are noted. Loss of fontanelle pulsatility occurs as intracranial hypertension worsens. Cerebral edema may be fatal despite a relatively compliant infant skull (**Chapter 15**).

AHT-associated skull fractures have patterns that cross suture lines, are numerous, and can be widely diastatic. AHT primary brain injury is compounded by secondary injury resulting from hypoventilation, hypoxia, and hypoperfusion. Neurogenic fever may occur within a few hours of injury. Seizures are common in abused infants, may be subclinical, and are often very difficult to control. These secondary insults lead to profound, progressive, global cerebral edema and ischemia.

Other etiologies for intracranial pathology must be considered in the differential diagnosis of child abuse. Appropriate diagnostic testing and treatment, including antibiotics and possibly dexamethasone, should be rapidly initiated even if bacterial meningitis is a remote possibility. Acyclovir should be considered if the clinical picture suggests encephalitis. Nontraumatic causes of intracranial hemorrhage can be further investigated with neuroimaging and evaluation of coagulation or metabolic disorders.

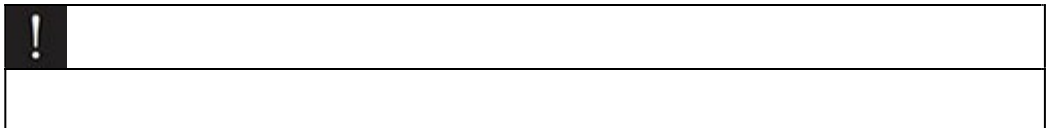
Lumbar puncture should be reserved for children who do not demonstrate obvious respiratory or hemodynamic abnormalities. In general, this procedure should not be performed in the emergency department during the initial evaluation and stabilization of critically ill children. Lumbar puncture carries significant risks, including cardiopulmonary arrest, herniation syndromes in the presence of intracranial hypertension, and spinal epidural hematoma if the child is coagulopathic. Thus, this procedure should not be performed until all such problems have been assessed or corrected.



Infants and young children who experience severe AHT are treated with airway stabilization, ventilator support, and management of increased intracranial pressure to mitigate secondary insults from hypoxia mass effect, cerebral edema, and seizures. Medical and surgical strategies are employed to decrease or control intracranial pressures (**Chapter 15**).

2. Retinal Hemorrhages

Retinal hemorrhages occur in 74% to 82% of cases of AHT; frequently, although not always, these accompany extra-axial hemorrhages and are the result of shear forces disrupting vulnerable tissue interfaces. The vitreous body is adherent to the retina in young children, and traction on this structure during rotational injury results in retinal hemorrhages occurring in multiple layers and extending to the periphery of the retina. This pattern is distinct from the focal abnormalities noted with increased intracranial pressure, sustained prolonged resuscitative efforts, and accidental traumatic brain injury. Medications that facilitate funduscopy, such as mydriatic drops, should be used with caution in children with evolving neurologic findings because it may complicate the early diagnosis of worsening cerebral edema by precluding use of pupillary reactivity as a harbinger of evolving intracranial hypertension. Retinal hemorrhages, although once considered a pathognomonic sign of abusive head injury, are also known to occur following cardiopulmonary resuscitation, metabolic diseases, coagulopathies, and other pediatric disease processes. While such findings may be suggestive of abusive injury, they must be considered in the context of the description of the mishap, the child's medical and developmental history, cultural factors, and the overall clinical picture.



Medications intended to facilitate funduscopy should be used with caution in children with evolving neurologic findings.

3. Blunt Abdominal Injury

Case Study

A 3-year-old boy is found on his doorstep by his grandmother. Emergency services personnel called to the home noted him to be tachycardic and hypotensive. He is resuscitated en route to the hospital. His abdomen is distended and tender to palpation.

Detection

– What is the possible diagnosis?

Intervention

– What needs to be done immediately to resuscitate this boy?

– How can the adequacy of resuscitation be determined as you proceed?

Reassessment

– What tests would be useful to evaluate the severity of the patient's condition?

– What radiographic studies are appropriate for evaluating the abdomen?

– What blood work should be obtained?

Effective Communication

– Where is the best place to manage this patient?

– Is a surgical consultation needed?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

Inflicted blunt abdominal trauma is most commonly seen in mobile children, typically toddlers and children of preschool age. These injuries usually have a delayed presentation and extremely high

associated morbidity. Although children in these age groups may also sustain inflicted head injuries, these differ from the pattern seen in young infants in that the injuries are usually due to direct or translational impact rather than rotational forces.

Abdominal injuries typically present many hours after they occur. These injuries follow AHT as the most common cause of death and severe morbidity in the abused child.

The combination of solid organ and hollow viscus injuries in the absence of corroborated high-velocity impact is highly suggestive of child abuse. The following organs are most commonly affected in inflicted abdominal injuries:

- Liver
- Spleen
- Kidney
- Pancreas
- Hollow viscus

Compressive forces may cause hematoma formation in the highly vascular duodenum. Although perforation may occur in any segment of the bowel, it is more common in the proximal jejunum due to that segment's relatively fixed position over the lumbar spine. Pancreatic contusion and transection may result in severe pancreatitis. Mesenteric disruption may compromise bowel perfusion, causing perforation. Hypovolemic shock may progress rapidly to uncompensated or even irreversible shock.

Much like inflicted severe head injuries, fatal abdominal injuries may occur without cutaneous evidence. Children may demonstrate abdominal tenderness, peritoneal signs, and distention on presentation. The diagnosis of abdominal injury may be missed in the presence of severe neurologic injury when bruising is absent. Given the life-threatening nature of abdominal injuries, they should be suspected and excluded in children presenting with other abusive injuries. Although computed tomography imaging readily demonstrates solid organ injury, hollow viscus injuries may escape detection until clinical deterioration occurs or a contrast study is performed.

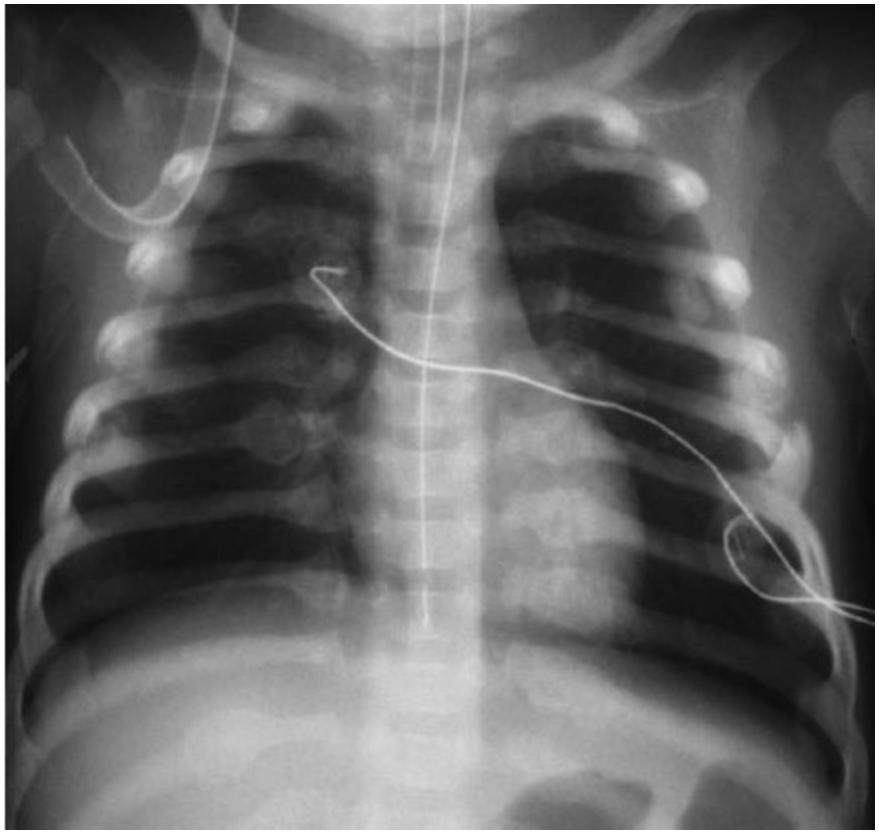
4. Other Injury Patterns

a. Skeletal Trauma

Fractures involving the posterior ribs, scapulae, sternum, and spinous processes, as well as the metaphyses in nonambulatory children, are often associated with child abuse. However, any fracture that does not appear to coincide with a child's developmental stage, the related findings, and the

reported mechanism of injury should raise suspicions of abuse and requires further evaluation. Posterior rib fractures in previously healthy infants indicate severe trauma to the skeletal system and, in the absence of a clear history of serious accidental trauma, are strongly linked to abuse. These fractures occur when a child is subjected to squeezing forces that cause disruption at the costovertebral junction. They are often not visible on plain radiograph until they are in the healing phase of callus formation (**Figure 11-4**).

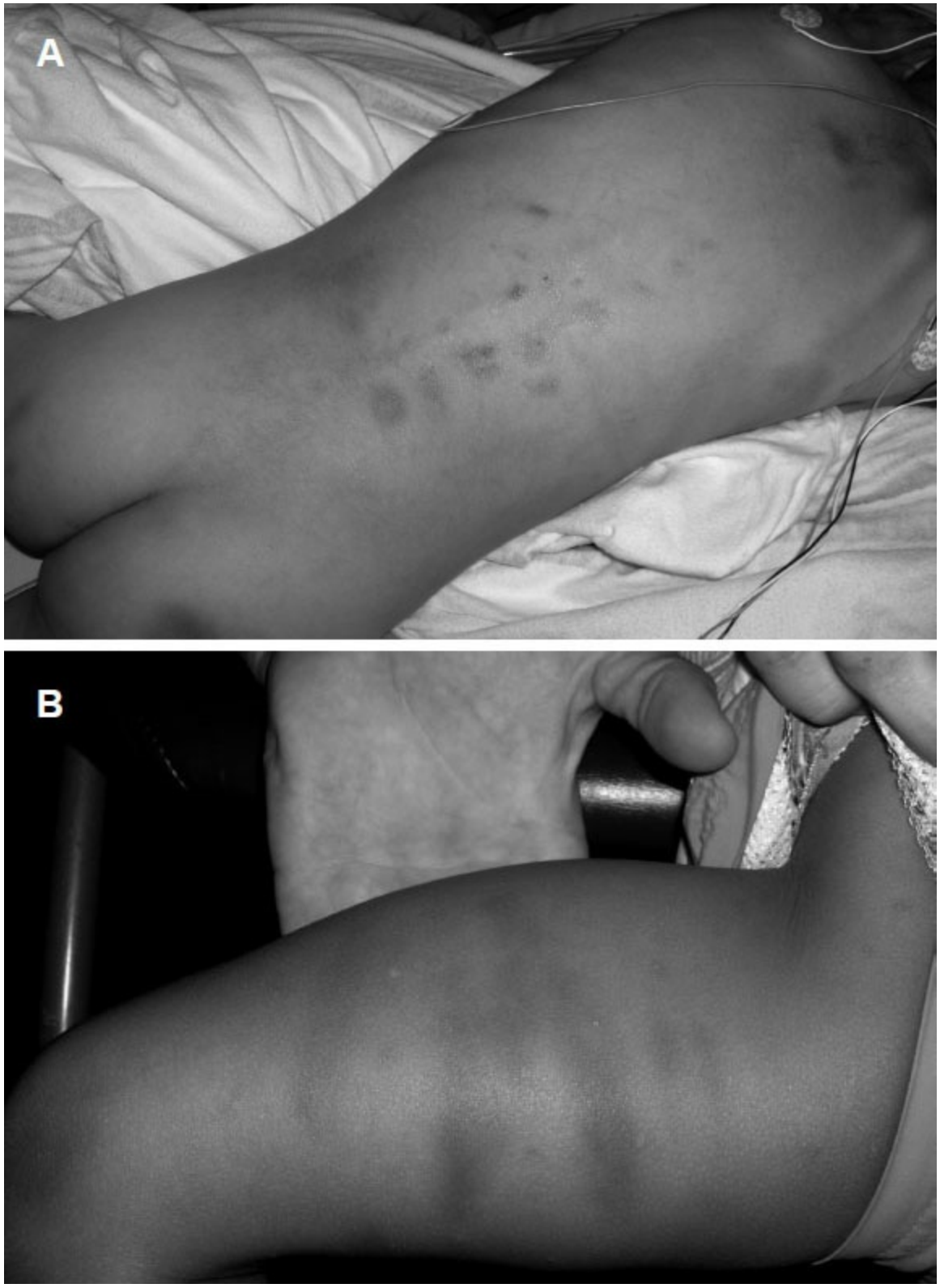
Figure 11-4. Numerous Healing and Acute Rib Fractures



b. Cutaneous Injury

Bruises that occur in nonambulatory children, are clustered in a pattern, occur away from bony prominences, and involve the abdomen, buttocks, back, face, and ears, with or without hands, should all be viewed with suspicion. Occasionally, cutaneous injuries have patterns that correlate with the object or implement used to inflict the injury (**Figure 11-5**). Bruises cannot be accurately dated by appearance, and medical professionals should be cautious in handling requests from investigative personnel to do so. Child abuse specialists may help determine the significance of cutaneous abnormalities in patients of specific cultural backgrounds. When the distribution of bruising does not match the history provided by family members, any medical and family history of easy bruising and bleeding should be obtained.

Figure 11-5. Patterned Abusive Bruising



A, Many scratches and bruises over the back can be identified.
B, Clearly patterned bruising is visible on this child's thigh.

c. Sexual Abuse

Not only is sexual abuse distressingly frequent and underreported, it also often goes undiagnosed until other injuries or even pregnancy prompt evaluation. Any patient presenting with perineal trauma with or without a history of sexual abuse should be carefully evaluated; if the findings are suspicious, a report should be filed and a child abuse expert should be consulted for further examination. In the absence of obvious perineal injury, patients presenting with clear disclosures of abuse should be reported and referred for expert evaluation.

Life-threatening abdominal or pelvic injury must be carefully excluded in any patient with perineal trauma.

When a child has a history of sexual contact within the last 72 hours, an evidence collection kit must be prepared using appropriate measures to ensure that the chain of evidence is not disrupted. Anal dilatation may be an incidental finding during the initial evaluation of deeply sedated, critically ill children and may mimic sexual abuse. This and any other troubling findings are best evaluated and excluded by a provider experienced in the evaluation of sexual abuse. The possibility of sexually transmitted disease must be addressed in any child presenting with suspected sexual abuse and post-exposure prophylaxis should be considered accordingly.

d. Asphyxiation

Asphyxiation injuries may be difficult to diagnose. Smothering may share some features of near-miss sudden infant death syndrome and presents as a global hypoxic-ischemic injury with varying degrees of neurological and multiorgan dysfunction. Careful questioning after the patient's initial stabilization may elicit a history suggestive of deliberate or accidental suffocation. However, the true etiology of the infant's collapse may never be determined.

e. Ingestions

Accidental ingestions are most common in children between 1 and 5 years of age. Ingestions in nonambulatory or preadolescent children should raise concern for abuse. All ingestions deemed to be accidental merit review of safety measures. Massive amounts of potentially lethal drugs may be forced upon children of all ages and may cause refractory hemodynamic instability with cardiac arrest. A comprehensive toxicology screen should be immediately obtained during the evaluation of any child with disturbed consciousness. Positive results for illicit drugs on screening drug tests should be confirmed to avoid reporting a potentially false-positive test. Prompt consultation with a toxicologist or a poison control center for optimal antidote and decontamination advice may be lifesaving (**Chapter 13**).

Caustic ingestions that are more severe than expected based on history or developmental stage may indicate that a child is living in a home where illegal drug use or manufacturing is occurring. This has become more common as the use of methamphetamines has increased.

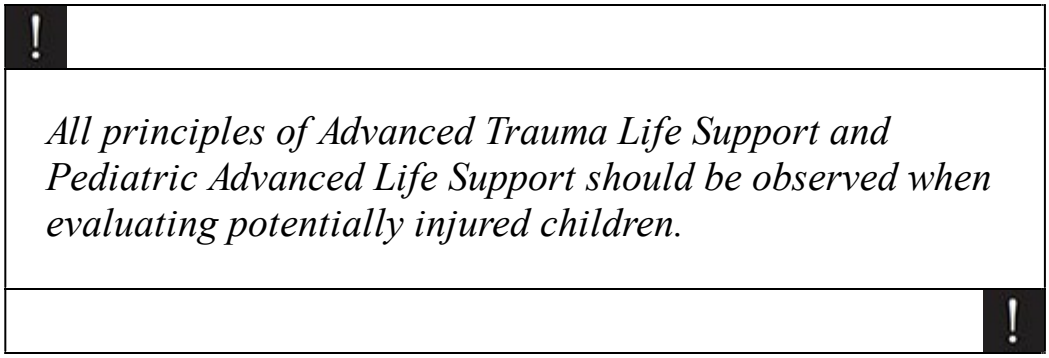
f. Medical Neglect

Medical neglect is yet another form of child maltreatment. The demands of caring for a chronically ill, technology-dependent child can become overwhelming. Substance abuse is a common comorbid factor in the negligent behavior of caregivers. Noncompliance with medical care may lead to near-fatal complications in children with diseases such as asthma, short bowel syndrome, and diabetes. Severe failure to thrive may present as a medical emergency when a child's weight is less than 70%

of the predicted weight for length. Such children are at significant risk for refeeding syndrome with resultant life-threatening electrolyte disturbances; thus, they require close monitoring during the initial stages of support.

III. ACUTE MANAGEMENT OF CHILD ABUSE

Optimal care of any acutely ill or injured child begins with complete initial assessment followed by frequent, serial reassessments of the patient's airway, breathing, circulation, and disability (neurological) status, followed by exposure and optimization of environment (*ABCDE*). A complete set of vital signs and a Glasgow Coma Scale score must be obtained on presentation and at frequent intervals thereafter. All principles of Advanced Trauma Life Support and Pediatric Advanced Life Support should be observed when evaluating potentially injured children. Specific stabilizing measures for the initial management of pediatric trauma are described in depth in **Chapter 9**.


!
All principles of Advanced Trauma Life Support and Pediatric Advanced Life Support should be observed when evaluating potentially injured children.
!

IV. DIAGNOSIS OF CHILD ABUSE

A thorough history must be obtained, with special emphasis on the caregiver's report of the mechanism of injury, and a complete physical examination must be performed. If possible, caregivers and the child should be interviewed separately as soon as possible after the incident to minimize the possibility that scripted accounts of the incident will cloud later reports. Historical details must be meticulously recorded, including but not limited to the following information: familial structure, child's previous medical condition, individuals with the child at the time of the incident, accurate description of the venue in which the reported incident occurred, precise timeline of any and all events associated with the incident, and actions taken by caregivers immediately thereafter. Given the variability in the presentation and history, clinicians should maintain a high index of suspicion. The physical examination must be performed with the child fully unclothed, taking care to avoid potential hypothermia. Any abnormality noted must be investigated with appropriate imaging and laboratory studies, and should be followed by serial clinical examinations as indicated.

Photographs of external findings, especially marks, bruises and superficial wounds, should routinely be obtained; such findings may change over time. Moreover, a picture will always provide a more accurate depiction than a drawing in the medical record. For photographs to have value, hospital policies must be followed and proper procedures used so that photographs identify the person depicted, date and time photo was taken, and the photographer.

Only emergent diagnostic imaging should be performed upon initial presentation. Nonemergent studies (eg, skeletal survey) should be obtained when they can be properly supervised by those familiar with child abuse. A poorly obtained study, even if repeated later, may present intractable problems for both the investigation and prosecution. Preliminary interpretation of emergently needed imaging should be obtained initially; all final interpretations and reports should be provided by a pediatric radiologist experienced in child abuse cases. This will minimize the risk of conflicting results or incorrect readings. Significantly, in some jurisdictions, nonemergent studies with an invasive component, such as those involving radiation, may require parental consent or court order if they are being obtained solely for forensic purposes. Finally, as with other types of traumatic injury, management involves a multidisciplinary team of pediatric subspecialists, including pediatric trauma surgeons, neurosurgeons, orthopedic surgeons, anesthesiologists, intensivists, child abuse pediatricians, and child maltreatment agencies, where available, should be consulted as soon as possible, as some injuries require urgent attention in the trauma suite.

A. Laboratory Tests

Initial laboratory studies are directed toward rapidly identifying end-organ injury and its sequelae, as well as guiding critical care management (Chapter 9). Studies particularly relevant to the diagnosis of child abuse are delineated in **Table 11-3** and should only be ordered after consultation with a child abuse pediatrician. Calcium, phosphorus, and alkaline phosphatase levels can be obtained after stabilization to screen for systemic conditions affecting bone mineralization. Specialized studies and consultation may be indicated if a possible metabolic or genetic bone disease etiology for fracture is suspected.

Table 11-3 LABORATORY STUDIES TO CONSIDER WHEN CHILD ABUSE IS SUSPECTED

Problem	Laboratory Studies to Consider[a]
Skeletal injury	Complete blood count Serum bicarbonate, creatinine phosphokinase Serum calcium, phosphorus, alkaline phosphatase 25-OH vitamin D
Bruising/bleeding	Complete blood count Coagulation studies Von Willebrand disease panel if bruising is unexplained or if family history of bruising/easy bleeding
Possible blunt-force abdominal trauma	Complete blood count with serial hematocrit Liver function tests Amylase and lipase Urine analysis
Altered level of consciousness	Bedside glucose and full electrolyte panel Toxicology testing Ammonia Liver function tests

[a]Other tests may be called for if the child's history, the family history, or the physical examination indicates other possible disease processes.

1. Computed Tomography Scan

Non-contrast computed tomography (CT) scan is the preferred study in children with suspected AHT. This study may be performed very rapidly and without sedation. Infants, toddlers, and children with decreased levels of consciousness should be immobilized for the scan in a manner that ensures airway patency, and with continuous monitoring during the study. Serial scans may be required in children found to have epidural hematomas, large evolving extra-axial collections, or a clinical picture of worsening cerebral edema.

A CT scan of the abdomen and pelvis will complement careful serial clinical examinations, but findings may be subtle in hollow viscus injuries. The role of ultrasonography is less clearly defined in this setting.

2. Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is not as sensitive as CT in identifying acute hemorrhage. It is also a more lengthy study, may require additional transport of fragile patients, and often requires monitored sedation or general anesthesia with the attendant risks. In general, initial neuroimaging should be limited to a non-contrast CT of the head unless concomitant spinal cord injury is suspected. In that case, emergent MRI may be considered. Monitored sedation or general anesthesia is necessary to avoid added hypoxic or ischemic insult. Once past the acute phase of care, MRI may be helpful to child abuse investigations in determining the age of findings, such as central nervous system bleeding and extra-axial fluid collections. There is increasing evidence that MRI may offer more support to dating the intracranial trauma utilizing signal analysis of resolving subdural bleeding on T1- and T2-weighted and fluid-attenuated inversion recovery sequences.

3. Skeletal Survey

A complete skeletal survey should be performed in children younger than 2 years of age when physical abuse is suspected. This should only be done under the supervision of a pediatric radiologist experienced in child abuse, and it is not considered an emergent study. Protocols which follow the guidelines developed by the Society for Pediatric Radiology should be employed. Anteroposterior views of all extremities should be obtained, including anteroposterior feet and posteroanterior hands. Such studies should ideally be performed in the radiology suite; however, the risk of transport for an unstable patient may preclude this, and portable films may be obtained initially in the pediatric ICU.

Acute fractures, particularly those involving the posterior ribs, may not be apparent on the initial skeletal survey in young children. Once the child's condition has stabilized, a bone scan is recommended in conjunction with the skeletal survey, because the radionucleotide tracer uptake in areas of increased bone turnover allows for identification of injuries that may go undiagnosed using standard radiographs. A repeat skeletal survey in 2 to 3 weeks to detect callus formation at fracture sites is an acceptable alternative to the bone scan in patients who cannot leave the pediatric ICU. Although rib and metaphyseal fractures may not require orthopedic intervention, long bone and spinal fractures may need stabilization.

The initial skeletal survey must include two-view skull, two-view chest with bone technique, anteroposterior pelvis and abdomen, and lateral lumbar spine images.

4. Funduscopy and Medical Photography

Funduscopy and photographic documentation by an ophthalmologist are important in establishing the extent and distribution of retinal abnormalities.

V. MEDICOLEGAL ASPECTS OF CHILD ABUSE

The management of child abuse may be extremely upsetting to the healthcare team. The physician and other immediate care providers must concentrate exclusively on the child's immediate medical stabilization and subsequent care. Involving social workers or child protection workers early is important. They can help gather vital information and can facilitate notification of the local child protection agency; all of these parties may act in concert with law enforcement to remove other vulnerable children from a dangerous environment.

Interactions with parents or other suspected perpetrators must remain civil. Remarks perceived as accusatory may hinder the child's medical care even if protective custody is not delayed. Unwise questioning may jeopardize the investigation.

The medical team's attention should be focused on medical stabilization of the child and support of the child's family. All aspects of the forensic investigation should be coordinated by social services and other child-advocacy professionals.

The role of the physician and other healthcare providers includes coordination of the child's medical care as well as complete, objective, and legible documentation of all relevant findings and events. Careful, clear documentation with appropriate medical imaging and photographs obtained during the child's hospitalization will be immensely helpful if a case proceeds to criminal trial. Complete, unbiased, accurate medical information must be presented clearly and understandably to facilitate judgments that are in the child's best interest, protect the innocent, and hold offenders accountable for their actions.

Every US state, Canadian province, and US or Canadian territory mandates that injuries suspicious for child abuse must be reported by the professional caring for the child. Failure to report suspected abusive trauma might enable repetitive, possibly fatal injury to the patient and endanger other children. Requirements for submitting a report—including whether such report must be personally submitted by the provider or can be done by another, such as a social worker, on the provider's behalf—often vary among jurisdictions. Thus, it is crucial to know the local statutes and regulations.

Key Points

"Abusive" Trauma: Diagnosis and Management

- Complete initial and serial assessments of airway, breathing, circulation, disability, and exposure and environment (*ABCDE*) are central to the management of any seriously ill or injured child.
- All principles of Advanced Trauma Life Support and Pediatric Advanced Life Support should be observed when evaluating potentially injured children.
- Initial evaluation includes a bedside glucose measurement and targeted laboratory and radiologic studies.
- Multidisciplinary consultation with pediatric surgeons, anesthesiologists, and pediatric intensivists should be obtained quickly.
- Children with abusive injuries typically present in a delayed fashion, complicating initial stabilization and eventual outcome.
- The team's efforts must be centered on the child's stability and comfort. Investigation of suspected abuse must be deferred to properly trained staff.
- Any negative factor, whether intrinsic to the child or to the caregiver, raises the risk of neglectful, abusive, and unintentional injury.
- Injury patterns may be suggestive, but are not reliably diagnostic, of abuse when compared with unintentional injury.

Suggested Readings

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PEDIATRIC EMERGENCY PREPAREDNESS

Objectives

- Detail current issues and deficiencies in federal and state disaster plans related to the care of special populations.
- Identify the differences between caring for children and caring for adults during a disaster.
- Review select important physiological differences between children and adults.
- Describe children's unique vulnerabilities in certain disaster situations.
- Outline the importance of maintaining the family unit during a disaster.
- Summarize how to adapt current disaster plans to meet the needs of children.

Case Study

A large fire broke out in a circus tent at the state fair. Over 450 individuals were present in the tent: about 300 adults and about 150 children. The children ranged in age from 6 months to 18 years. The smoke and chemicals caused both respiratory distress and failure, loss of consciousness, and burns. Of the 150 children exposed, 60 require tertiary level care. The surrounding communities have 30 pediatric ICU beds located within a 30-minute transport time, 10 local pediatric critical care physicians, and 10 local emergency department physicians who have experience in treating pediatric patients. Sixty adult ICU beds are available to care for the adult victims.

Detection

– What factors make children more susceptible to respiratory and airway injury?

– What is the best way to triage the large number of pediatric victims at the scene?

– How do you handle overcrowding from ambulance arrivals and frantic parents who drive directly to the hospital with their children covered in soot?

– When every minute counts, what would be the most effective way to triage a large number of pediatric victims during a disaster?

Intervention

– Is it known if the injuries are airway injury, carbon monoxide poisoning, or burns?

– Were additional chemical toxins released that may need an antidote?

Reassessment

– Is the local healthcare system equipped to manage a large number of pediatric patients?

– What is the next step if the number of pediatric-trained critical care, emergency medicine, or acute care personnel is insufficient?

– Can resources be pulled from the adult teams to manage the older children?

– Will adult patients also utilize the hospital resources?

Effective Communication

– Where will the triage occur on-site?

– Where will the triage occur off-site?

– Who is the onsite person communicating within the hospital and with the local emergency teams?

Teamwork

– Who should be directing where patients should be allocated?

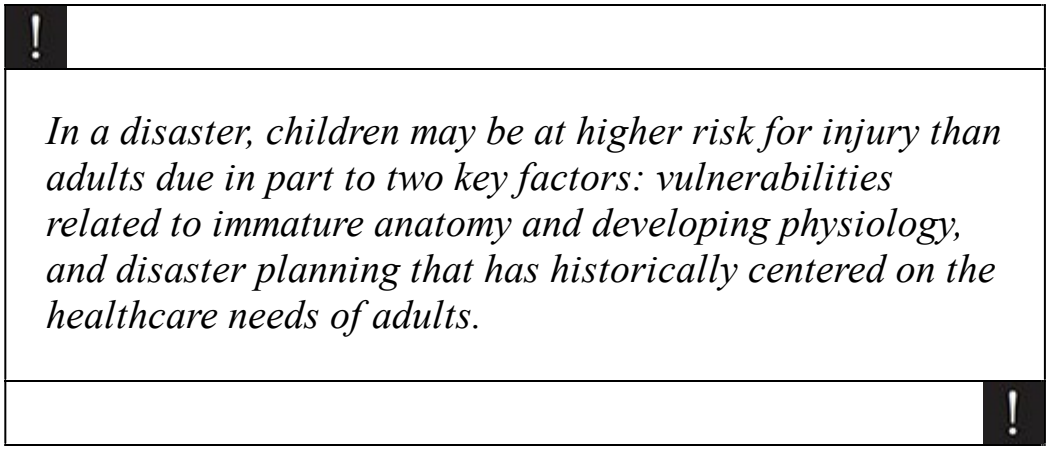
I. INTRODUCTION

Preparation matters in disasters involving pediatric patients, because children may be at higher risk for injury than adults because of two key factors: 1) vulnerabilities related to their immature anatomy and developing physiology, including unique psychosocial factors (eg. mental health needs, school environments, and childcare centers); and 2) a disaster planning system that has historically centered on adult healthcare needs. Knowledge about multiple casualty incidents (MCIs), mass casualty events (MCEs), terror injuries, and the disaster response has come primarily from military experience. For this reason, planning for disasters, including acts of terrorism, has focused almost exclusively on the adult population.

While the operational definition of a disaster is an event in which the needs of those affected outstrip the resources available for an adequate level of care and support, experts define an MCI as an incident involving ≥5 victims. In most circumstances, the resources available to care for these patients will be strained, but not overwhelmed, and the effects on the healthcare system will usually be limited to the closest hospitals. By contrast, an MCE involves ≥20 victims and may destabilize the healthcare system(s) serving the entire region. In reality, few hospitals are prepared to accept such a high number of patients in a limited period, and chaos typically ensues. As a general rule, emergency department organization begins to break down when a hospital's casualty intake begins to exceed 7 critically ill or injured patients per hour, at which point the critical mortality rate—which correlates directly with the overtriage rate and is the key metric of performance during an MCE—begins to

exceed a baseline of approximately 10%.

In 2010, almost a quarter of the total US population was younger than 18 years. Because the pediatric population is generally healthy compared with the adult population, peacetime medical systems allocate resources primarily to adults. That leaves only a few pediatric trauma centers to address critically injured children and limits the ability of community health systems to promote healthcare and disease-prevention strategies grounded in a well-child paradigm. Therefore, a disaster that affects the US population will take a higher toll among the vulnerable pediatric population and require more resources than are available. In previous large-scale natural disasters in the United States between 1992 and 2001, pediatric patients (average age of 4 years) represented one-third of all patients treated by disaster teams. In a recent evaluation of the hospital surge capacity in New York State, area hospitals were found to have approximately one-half of the pediatric inpatient surge capacity recommended by US federal disaster-planning authorities and exceeded the recommended surge capacity for adult inpatients. Unlike the established US trauma system, nonpediatric trauma centers—indeed, hospitals in general—must be prepared to receive and triage ill and injured children in a disaster. One study indicated that in a major pandemic, the current hospital capacity for the expected number of critically ill or injured children would not be sufficient, even if capacity was increased by following recommendations for pediatric emergency mass critical care. Thus, hospitals that normally care for a limited number of low-acuity children will need to supplement children's hospitals that have reached maximum surge capacity, and all hospitals will require input from pediatric medical experts to provide care for pediatric patients in a disaster.


!
In a disaster, children may be at higher risk for injury than adults due in part to two key factors: vulnerabilities related to immature anatomy and developing physiology, and disaster planning that has historically centered on the healthcare needs of adults.
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Pediatric care is a specialty, and only a small number of acute care, critical care, and emergency care providers are trained to assess, diagnose, and expeditiously treat severely injured children. Consequently, when a disaster strikes, generalist care providers whose knowledge of pediatrics may be limited will be called upon to care for critically ill and injured children, while pediatricians who may not have been trained to handle such situations will need to take leadership roles in treating pediatric patients with critical conditions. Given that pediatric emergency skills diminish if not used or practiced, regular exercises in disaster response are critical. Equipment must be available in a range of sizes to accommodate children of different age, development, weight, and size. Although most large medical centers may have appropriate supplies, specialty equipment may not be available at hospitals that do not routinely treat and triage children. Similar deficiencies in equipment and training may also exist among disaster medical assistance teams.

In the interest of efficiency, US federal and regional medical supply stockpiles have adopted a one-

size-fits-all approach. This is problematic because adult drug dosages may prove harmful in the younger population. Unfortunately, pediatric dosing requirements may be difficult to achieve due to how medications are stored, packaged, and prepared. The US Strategic National Stockpile of medical supplies has been modified so that a 12-hour push pack designed to supplement local supplies can be immediately dispatched after an emergency. The push pack contains medications that are approved by the US Food and Drug Administration (FDA) for use in children, but many of those medications arrive in adult-dose aliquots. Moreover, antidotes for chemicals used in terrorist attacks are not approved for children because significant questions remain regarding effective, nontoxic pediatric dosages. To have sufficient quantities of appropriate medicines available for children's needs in a disaster, altered standards of care would need to include dosage adjustments, changes to expiration dates, and guidelines for the use of adult-specific medications in children.

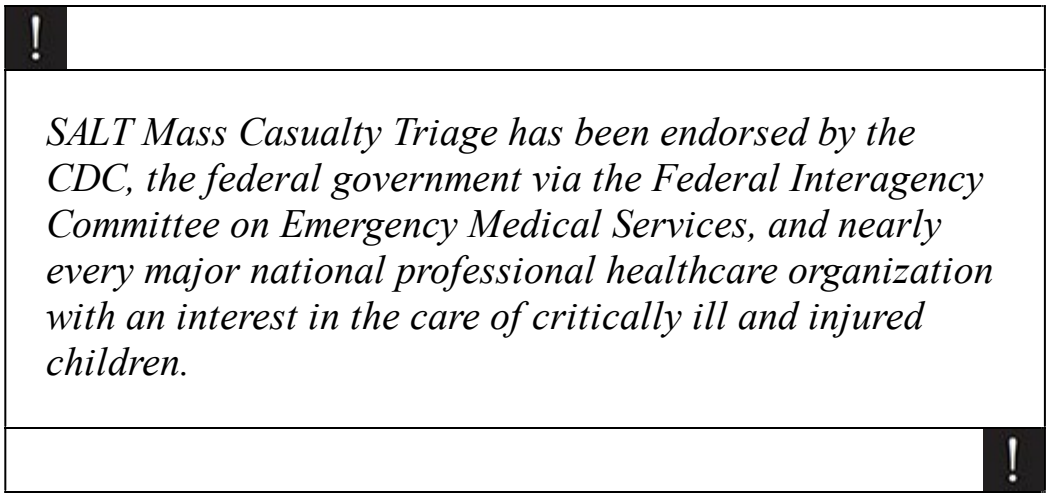
Preparation is the key to an effective disaster response. In 2010, the US Centers for Disease Control and Prevention (CDC) created the Task Force for Pediatric Emergency Mass Critical Care to address the needs of critically ill and injured children in disasters. The report of this task force was published as a supplement to the academic journal *Pediatric Critical Care Medicine* in November 2011. It calls for significant increases in surge capacity—a doubling of pediatric ICU beds and a tripling of pediatric critical care medicine capability— utilizing alternate sites within the hospital that can be temporarily outfitted, staffed, and equipped to meet the needs of critically ill and injured children.

II. TRIAGE

During a disaster when resources are limited, hospitals must have rapid and reliable triage systems to prioritize the care of patients by severity and resource availability. In MCIs, where resources are strained but not overwhelmed, it is reasonable to utilize standard triage practices, whereby the most critical patients are prioritized with respect to the threat posed to the airway, breathing, circulation, and disability. As an example, a patient with an immediate life threat to the airway would be treated before a patient with an immediate life threat to breathing. However, in MCEs, given the large number of patients who must be rapidly triaged and treated, "reverse" triage is utilized. At the scene, reverse triage lets the least critical patients be identified first and tagged appropriately (green-tagged patients require minor care, yellow-tagged patients require delayed but not immediate care), allowing them to be temporarily bypassed to permit resources to be directed initially to those most likely to benefit from lifesaving interventions (red-tagged patients require immediate care)—excluding those for whom critical resources are not initially adequate to the need (grey-tagged patients require expectant care) and those who are unsalvageable (black-tagged patients are dead or expected to die under any circumstance). In the hospital, reverse triage takes the form of a rapid discharge plan, whereby stable patients undergo early discharge from the emergency department or critical care unit to less acute environments of care, thereby making room for patients in greater need of emergency and critical care resources. The overarching principle is to provide the greatest good for the greatest number of patients.

Prehospital triage systems are applied during a disaster when the main goal becomes allocating limited resources quickly to salvageable patients. Without an effective triage system, any emergency department can be quickly overwhelmed during an MCI or MCE. There are many prehospital triage

systems, but the most commonly used are START (Simple Triage and Rapid Transport), JumpSTART, and SALT (Sort, Assess, Lifesaving Interventions, Treatment and Transport). Although START likely remains the most commonly used prehospital triage system in the United States, it has significant limitations. JumpSTART was created to address some of these limitations with respect to children, but it is has not been widely adopted. The CDC empaneled an expert technical advisory group in an effort to unify prehospital triage systems across the country. The result was SALT Mass Casualty Triage (**Figure 12-1**), which takes into account pediatric physiology. This system has been endorsed not only by the CDC and the rest of the federal government via the Federal Interagency Committee on Emergency Medical Services, but also by nearly every major national professional healthcare organization with an interest in the care of critically ill and injured children. Despite this wide acceptance, SALT has not been adopted in many locales. Pediatric critical care providers must be familiar with the prehospital triage system that is utilized in their community.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SALT Mass Casualty Triage has been endorsed by the CDC, the federal government via the Federal Interagency Committee on Emergency Medical Services, and nearly every major national professional healthcare organization with an interest in the care of critically ill and injured children.
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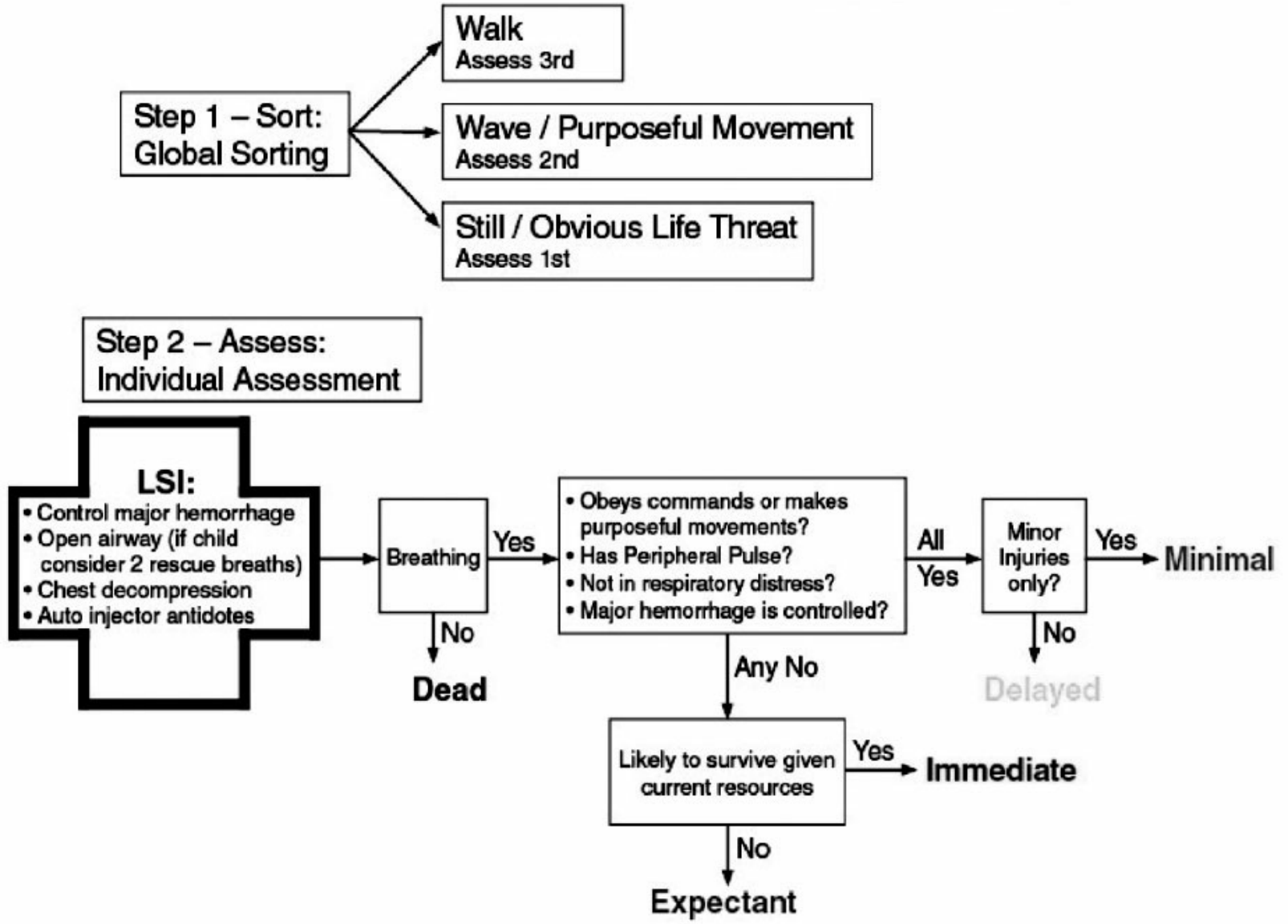
Any pediatric prehospital triage methodology should:

1. Consider developmental stages
2. Consider physiologic variations in vital signs.
3. Take into account that respiratory collapse often precedes cardiovascular compromise.
4. Understand that children may not obey verbal commands.

Figure 12-1. SALT Mass Casualty Triage

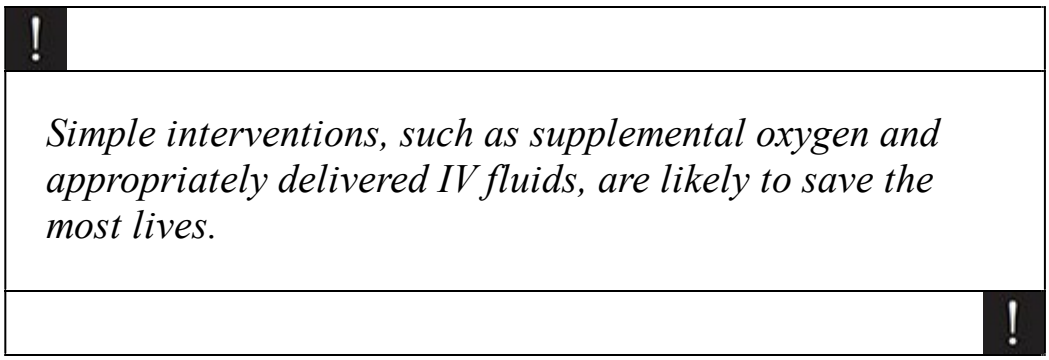


LSI, lifesaving intervention.
Reprinted with permission from Lerner EB, Schwartz RB, Coule PL, et al. Mass casualty triage: an evaluation of the data and development of a proposed national guideline. *Disaster Med Public Health Prep*. 2008;2(Suppl 1):S25-S34.Permission pending

In the SALT system, step 1 is global sorting, which facilitates rapid identification of patients requiring minor or delayed treatment. Step 2 is the initial assessment or rapid identification of the more seriously injured patients for lifesaving interventions (hemorrhage control, airway opening, chest decompression, auto-injector antidotes). If the patient is not breathing after these interventions, he or she is considered dead. However, the breathing patient is briefly assessed with respect to the ability to obey commands or make purposeful movements, the presence of a peripheral pulse or respiratory distress, and control of major hemorrhage. If any of these assessments is abnormal, and the patient is deemed unlikely to survive given current resources, he or she is a candidate for expectant care. Those likely to survive given current resources are candidates for immediate care. By contrast, if all of these assessments are normal, and the patient has more than minor injuries, he or she is a candidate for delayed care, while those with only minor injuries are candidates for minimal care, which typically is provided after the needs of other patients have been addressed.

While START, JumpSTART, and SALT are examples of primary triage systems in the prehospital setting, the purpose of secondary triage is to determine where the patients will be ultimately placed in

the hospital (ie, emergency department, inpatient unit, operating suite, postanesthesia care unit, or critical care unit) or to decide if the patient must be stabilized and transferred to a facility with greater capacity or capabilities. Thus, secondary triage helps to utilize limited resources in the most effective way during a disaster. Several clinical scoring systems have been developed to predict the outcomes of pediatric ICU patients, including the Modified Sequential Organ Failure Assessment (mSOFA), Pediatric Risk of Mortality (PRISM III), Pediatric Index of Mortality (PIM 2), Pediatric Multiple Organ Dysfunction Score (P-MODS), and Pediatric Logistic Organ Dysfunction (PELOD). Several states have proposed criteria for secondary triage with respect to ventilator allocation in pandemic influenza. However, no validated score addresses the potential outcomes of children critically injured or ill as a result of a disaster. The difficult issue of rationing based on predictions of mortality in the pediatric population is made even more difficult by the special emotional bonds adults share with children. Both under- and over-triage practices will cause significant mortality and morbidity during a disaster. That said, simple interventions, such as supplemental oxygen and appropriately delivered IV fluids are likely to save the most lives.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Simple interventions, such as supplemental oxygen and appropriately delivered IV fluids, are likely to save the most lives.
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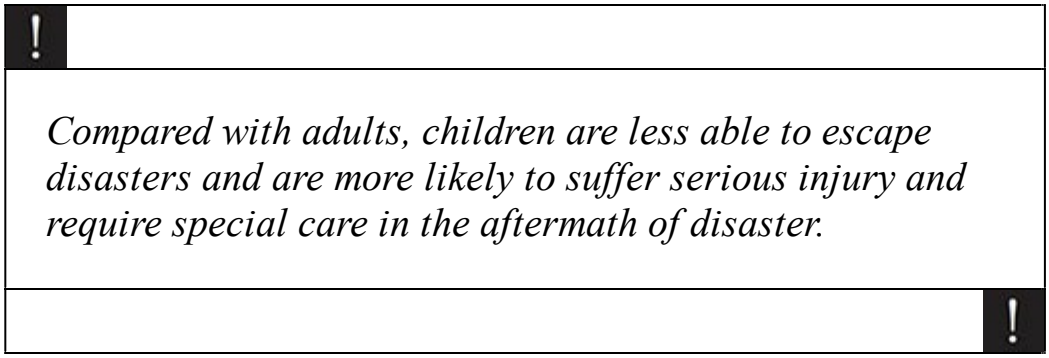
III. INCIDENT COMMAND SYSTEMS

After-action reports following major disasters consistently emphasize the vital role of intra- and interorganizational communication in managing the resources available and the untoward consequences of communication failure on the disaster response. In 2003, US Homeland Security Presidential Directive-5 established the National Incident Management System, a key component of which is the local Incident Command System activated by the regional Emergency Operations Center in the event of an MCE or disaster. The Incident Command System has 3 key elements: 1) unity of command, which prescribes that individual responders receive formal orders from, and give formal reports to, a single individual, so as to maintain the chain of command; 2) span of control, which limits the number of responders (5-7) who may be supervised by a single individual, keeping the volume of information that must be transmitted at a manageable level; and 3) clarity of text, which obligates individual responders to use plain language when transmitting information, because the healthcare, public health, and public safety agencies involved in a disaster response rarely share the same professional jargon. As such, it is apparent that the Incident Command System is also an "incident communication system" and is the glue that binds responders from the participating agencies into a single force.

IV. UNIQUE ANATOMIC AND PHYSIOLOGIC VULNERABILITIES IN

CHILDREN

Because the needs of the pediatric population tend to be overlooked in disaster planning, healthcare leaders need to revisit disaster plans to ensure that they include appropriate care for pediatric patients. Compared with adults, children are less able to escape disasters and more likely to suffer serious injury and require special care in the aftermath of disaster. Even in day-to-day situations, age-related anatomic and physiological differences between children and adults can complicate the work of providers without experience in pediatric assessment and triage (**Appendix 1**). Substantial differences in anatomic maturity, physiologic development, and cognitive abilities also may increase the impact of a disaster on children.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Compared with adults, children are less able to escape disasters and are more likely to suffer serious injury and require special care in the aftermath of disaster.
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A. Respiratory Vulnerabilities

Because children are closer to the ground than adults, they are at increased risk for severe airway injury caused by chemical agents that are heavier than air, such as chlorine and ammonia. A child's rapid respiratory rate and small body surface area increase the intake of agents that act on the respiratory system and the metabolic effect of those agents. Inhalational agents that cause mucosal irritation and airway edema may have more impact on children than on adults. Airway resistance increases proportionately to the fourth power of the airway's radius, so a small amount of edema or mucus can obstruct a child's airway and lead to severe respiratory compromise and death (**Chapter 2**).

In addition, children's short stature may make them more vulnerable to drowning during flooding. Over 90% of pediatric deaths during the T-ohoku earthquake and tsunami were attributed to drowning. Aspiration of water contaminated with bacteria, soil, and chemicals, as seen in victims of near-drowning episodes, will most likely predispose them to develop pneumonia and pneumonitis. This is referred to as *tsunami lung* and is well described in both the 2004 tsunami in Southeast Asia and the 2011 earthquake in Japan.

The compliant chest wall and relatively underdeveloped intercostal muscles in infants and young children require the diaphragm to play a greater role in ventilation than it does in adults. The work of breathing may consume a large portion of available energy and predispose children to respiratory distress and failure. A child's oxygen demand per kilogram of body weight is twice that of an adult (6-8 mL/kg/min as opposed to 3-4 mL/kg/min), so hypoxemia will occur rapidly with inadequate alveolar ventilation.

Compared with adults, a child's tongue is somewhat larger in proportion to the oropharynx and may obstruct the airway in response to a minimal amount of lethargy. The trachea is flexible and narrow, which permits the airway to kink with flexion (as in inappropriate resuscitation techniques). A child's airway anatomy allows only a narrow range of positions to open the airway, and airway occlusion with secretions or edema is common.

B. Circulatory Vulnerabilities

A child's circulating blood volume represents a larger percentage of total body weight than it does in adults (70-80 mL/kg vs 55-65 mL/kg). However, due to a child's relatively small absolute total blood volume, what may be seen as minor bleeding for an adult can be substantial blood loss in a child. Relative to body surface area, a child has a smaller fluid reserve and immature kidneys, which combine to leave the child more prone to hypovolemia and hypovolemic shock. From a global perspective, diarrhea leading to hypovolemia and shock is responsible for 17% of all deaths of children younger than 5 years. In the aftermath of a disaster, contaminated food and water supplies may contribute to severe gastrointestinal illness, causing significant morbidity and mortality rates in exposed children.

Children have the ability to compensate for volume loss by increasing the heart rate. They also have a strong vascular motor response, which preserves preload and afterload. Tachycardia and vasoconstriction can mask the signs of shock, like hypotension, until a child is nearly moribund. Uncompensated shock progresses more rapidly to cardiopulmonary failure and arrest in children than in adults.

C. Neurologic Vulnerabilities

Children suffer a different constellation of injuries caused by trauma. A child's head is larger relative to body size than an adult's, and the cranial bones and blood vessels are thinner relative to body size. This, coupled with poor muscle strength in the neck, predisposes children to head and neck trauma. Fractures of the C1 and C2 vertebrae account for 70% of neck fractures in the pediatric population and only 15% in the adult population. Severe shock forces or a high-velocity explosion may predispose children to severe debilitating injuries that would result in placing them in a low-priority category during triage.

Myelinization is incomplete in small children, which makes assessment of neurological injury and recovery potentially more difficult. Immaturity of esterase enzymes, including acetylcholinesterase, may make small children vulnerable to pesticides and nerve agents. Children are also more prone to seizures than adults, so smaller doses of toxic agents are likely to have greater negative consequences in terms of both morbidity and resource utilization.

D. Musculoskeletal Vulnerabilities

A child's liver and spleen are relatively large and not as well contained within the rib cage as in adults, so these organs are vulnerable to blunt trauma. Because a child's ribs are flexible, they rarely

break, and the force of an impact is transmitted to the underlying and unprotected vascular organs, which increases the risk of internal injury with hemodynamically significant hemorrhage.

A child's body surface area is large in relation to weight, and the layers of skin are thinner than in adults. Both factors place children at increased risk for significant chemical and thermal burns and for hypothermia in response to environmental exposure. They also have consequences for treatment: large-volume, unheated adult decontamination showers will likely result in significant hypothermia in a small child.

E. Developmental and Psychological Vulnerabilities

Children are more vulnerable during a disaster simply because they are often unable to recognize or escape danger. They may even be drawn toward dangerous situations because of their inherent curiosity. Paradoxically, they may flee rescuers due to the often frightening appearance of people in personal protective equipment (**Figure 12-2**). Small children are often unable to verbalize what they have experienced, which may confuse or mislead responders and result in inappropriate care. With their larger body surface areas, children will also lose heat more rapidly than adults, which may lead to hypothermia.

Figure 12-2. Rescuer in Protective Clothing

It may be difficult to decontaminate and evacuate children because they cannot be expected to follow group directives and require continuous psychological support. They may have adverse reactions to caregivers, owing to fear of strangers or pain. Certain procedures, such as open wound management,

will be time-consuming in frightened children. Members of the care team with experience in calming and reassuring children should be placed in charge of such situations. Because they are unable to care for themselves, many uninjured children may wander about and require shelter.

V. TREATING CHILDREN'S DISASTER-RELATED INJURIES AND ILLNESSES

Despite their greater vulnerabilities, children with acute injuries or illnesses are more likely than adults to respond to rapid and efficient medical care. In fact, most care rendered to children in a disaster will be supportive; as a result, the benefits from adequate preparation will be significant. Simple interventions, such as supplemental oxygen and IV fluids, can save many injured children; however, certain exposures in a disaster will require the use of specific therapies. This section identifies these specific needs in a variety of disaster situations.

A. Exposure to Chemical Agents

Because of their rapid respiratory rates, children are vulnerable to injury from chemical agents in gaseous form. Gases that are heavier than air, like chlorine and ammonia, may accumulate in the small child's breathing space more rapidly than in an adult's. Nerve agent intoxication may manifest differently in children than in adults. During acetylcholine overload caused by nerve agents, children may be prone to dehydration and shock because of fluid loss due to gastrointestinal secretions. Children are also more likely than adults to have seizures during a cholinergic crisis.

Antidotes to nerve agents and pesticides are often available in auto-injectors, but both the dose and the needle length may be inappropriate for children. Neither atropine nor pralidoxime has been extensively tested in children, and newer antidotes have not been tested in children at all. In cases of severe exposure, if adult-size DuoDote auto-injectors (Meridian Medical Technologies, Inc., Columbia, MD) are the only source of antidote, they should be used in even the smallest children. Dosing recommendations for anticholinesterase exposures are given in **Table 12-1**.

Table 12-1 ANTIDOTE DOSING FOR ANTICHOLINESTERASE EXPOSURE IN CHILDREN

		DuoDote Auto-Injector Dosing for Severe Symptoms		
Agent	**Dose and Management**	**3-7 years or 13-25 kg**	**8-14 years or 26-50 kg**	**>14 years or >50 kg**
Atropine	0.05 mg/kg IV or IM for moderately severe symptoms, up to 0.1 mg/kg IV or IM in clear cholinergic crisis every 2-5 min as needed for marked secretions, bronchospasms, hypoxia, respiratory compromise	1 injector delivers 0.08-0.13 mg/kg	2 injectors deliver 0.08-0.13 mg/kg	3 injectors deliver <0.11 mg/kg

Pralidoxime chloride	20-50 mg/kg IV or IM to a maximum 1 g IV or 2 g IM; may repeat every 30-60 min for weakness or high atropine requirement up to 2000 mg/h	1 injector delivers 24-46 mg/kg	2 injectors deliver 24-46 mg/kg	3 injectors deliver <35 mg/kg
Diazepam	0.05-0.3 mg/kg IV			
Lorazepam	0.1 mg/kg IV or IM			
Midazolam	0.1-0.2 mg/kg IV or IM			

IM, intramuscular
Classified using data from *America's Children: Key National Indicators of Well-Being. 2017*. Federal Interagency Forum on Child and Family Statistics; 2017. http://www.childstats.gov/americaschildren/demo.asp. Accessed April 2, 2018. Rotenberg JS, Newmark J. Nerve agent attacks on children: diagnosis and management. *Pediatrics*. 2003;112:648-658.

With their thin skin, children may be more vulnerable to the effects of vesicants, placing them at increased risk for systemic absorption. Given their high ratio of body surface area to weight, they may lose a larger percentage of their skin than adults when exposed to the same volume of chemical agent. Large chemical burns increase the risk for dehydration, hypovolemic shock, and hypothermia.

Vesicants, such as mustard agents, that are delivered in explosive blasts may more readily cause occlusion of children's small airways. Decontamination may be more difficult because children cannot follow directions and may need to be held throughout the procedure. High-pressure washing may injure small children, and cold-water washing may cause hypothermia and shock. Children are best decontaminated by attendants using warm water, but such a labor-intensive approach may not be practical during a disaster without meticulous advance planning and dedicated teams of responders at the scene.

B. Radiation Exposure

The same factors that increase a child's risk of inhaling chemical agents also increase the risk of inhaling radioactive gases. Radiation-induced cancers occur more often in children than in adults exposed to the same dose. In utero exposure to radiation can cause intellectual disability and other congenital anomalies. Breast milk is quickly contaminated with radiation, so infants of exposed mothers may not be able to breastfeed. Treatment with potassium iodide should be initiated only if the local public health agency declares that radioiodides are present in the environment. Children ≤17 years of age, pregnant women, and lactating women are treated at lower exposure levels than those for adults. To protect the thyroid from radioactive iodine, treatment with potassium iodide is indicated if the exposure projected by government sources is >0.05 Gy (>5 rad). See **Table 12-2** for recommended dosages for children ≤17 years and pregnant and lactating women. Neonates treated with potassium iodide should have the thyroid function checked between 2 and 4 weeks after a single dose.

Table 12-2	POTASSIUM IODIDE DOSING FOR CHILDREN ≤17 YEARS AND PREGNANT OR LACTATING WOMEN

Infants (including breastfed infants)
Infants have the highest risk of developing thyroid cancer after being exposed to radioactive iodine. The US Food and Drug Administration (FDA) recommends that all infants, including breastfed infants, be given the dosage of potassium iodide recommended for infants. Infants (particularly newborns) should receive a single dose of potassium iodide; additional doses may lead to problems with normal development. Other protective measures should be used. In cases where >1 dose is necessary, medical follow-up may be indicated.

- Newborns should be given 16 mg (¼ of a 65-mg tablet or ¼ mL of solution). This dose is for both nursing and nonnursing newborns.
- Infants and children between 1 month and 3 years of age should take 32 mg (½ of a 65-mg tablet or ½ mL of solution). This dose is for both nursing and nonnursing infants and children.

Children
The FDA recommends that all children ≤17 years of age internally contaminated with (or likely to be internally contaminated with) radioactive iodine receive potassium iodide treatment, unless they have known allergies to iodine (contraindications).

- Children between 3 and 17 years of age should be given 65 mg (one 65-mg tablet or 1 mL of solution). Larger children (≥150 lbs) should take the full adult dose, regardless of their age.

Pregnant and Lactating Women
Because all forms of iodine cross the placenta, the FDA recommends that pregnant women should take potassium iodide to protect the growing fetus. Pregnant women should take only 1 dose following internal contamination with (or likely internal contamination with) radioactive iodine. Because all forms of iodine can enter breast milk, the FDA recommends that lactating women who are breastfeeding should take only 1 dose of potassium iodide if they have been internally contaminated with (or are likely to be internally contaminated with) radioactive iodine. They should be prioritized to receive other protective action measures.

- Pregnant women and lactating women who are breastfeeding should take the adult dose of 130 mg (one 130-mg tablet or two 65-mg tablets or 2 mL of solution).

C. Exposure to Biological Agents

The signs and symptoms of exposure to biological agents in children will be similar to those seen in adults. Fluoroquinolones and tetracycline (both used for anthrax and plague exposure) are contraindicated in growing children. See **Table 12-3** for acceptable alternative treatments.

Table 12-3 TREATMENT FOR EXPOSURES TO BIOLOGICAL AGENTS

Infectious Agent	Pediatric Prophylaxis	Pediatric Therapy	Notes
Inhalational anthrax	Ciprofloxacin 10-15 mg/kg PO every 12 h (maximum 500 mg/dose) for 60 days or doxycycline 2.2 mg/kg PO every 12 h (maximum 100 mg/day) for 60 days	Ciprofloxacin 10-15 mg/kg IV every 12 h (maximum 400 mg/dose) or doxycycline 2.2 mg/kg IV every 12 h (maximum 100 mg/day) and clindamycin 10-15 mg/kg IV every 8 h and penicillin G 250-600 U/kg/d divided every 4 h	Patients who are clinically stable can be switched to a single oral agent (ciprofloxacin or doxycycline) to complete a 60-day course of therapy
Cutaneous anthrax		Penicillin V 25-50 mg/kg/d PO divided every 6 h or amoxicillin 40-80 mg/kg/d PO divided every 8 h or ciprofloxacin 10-15 mg/kg PO (maximum 1 g/day) every 12 h or doxycycline 2.2 mg/kg PO every 12 h (maximum 100 mg/d)	
Gastrointestinal	Same as inhalational		

anthrax	anthrax		
Plague	Gentamicin 2.5 mg/kg IV every 8 h or doxycycline 2.2 mg/kg IV (maximum 200 mg/d) or ciprofloxacin 15 mg/kg IV	Gentamicin 2.5 mg/kg IV every 8 h or streptomycin 15 mg/kg IM every 12 h or ciprofloxacin 15 mg/kg IV every 12 h (maximum 400 mg/dose) doxycycline 2.2 mg/kg IV every 12 h (maximum 200 mg/d) or chloramphenicol 25 mg/kg every 6 h (maximum 4 g/d)	Chloramphenicol may be preferable in plague meningitis; use in or young children may be associated with serious side effects
Tularemia		Same as for plague	
Botulism		Supportive care; antitoxin from CDC	
Brucellosis		TMP/SMX 30 mg/kg PO every 12 h for 6-week course and rifampin 15 mg/kg every 24 h or gentamicin 7.5 mg/kg IM every day for 5 days	

PO, by mouth; IM, intramuscular; CDC, Centers for Disease Control and Prevention; TMP/SMX, trimethoprim and sulfamethoxazole

Classified using data from *America's Children: Key National Indicators of Well-Being. 2017*. Federal Interagency Forum on Child and Family Statistics; 2017.
http://www.childstats.gov/americaschildren/demo.asp. Accessed April 2, 2018.

Children may be at increased risk for infection with smallpox because they have no immunity to the virus, whereas adults who were vaccinated as children may be protected. Children are efficient vectors for infectious agents spread by droplets and may be affected in numbers out of proportion to the population. Due to their increased susceptibility to respiratory failure, in an epidemic of respiratory illness, children may have an increased need for mechanical ventilation and be at increased risk for fatal outcomes. Oseltamivir phosphate, which may be effective against influenza when used within the first 48 hours of symptom onset, is not approved for use in newborns younger than 2 weeks of age. Moreover, there is very little research to support the use of oseltamivir before, during, or after a pandemic. Zanamivir, another neuraminidase inhibitor approved for pediatric use in those as young as 7 years, has a more promising profile than oseltamivir for seasonal as well as pandemic flu. Those strains have not shown any resistance to zanamivir.

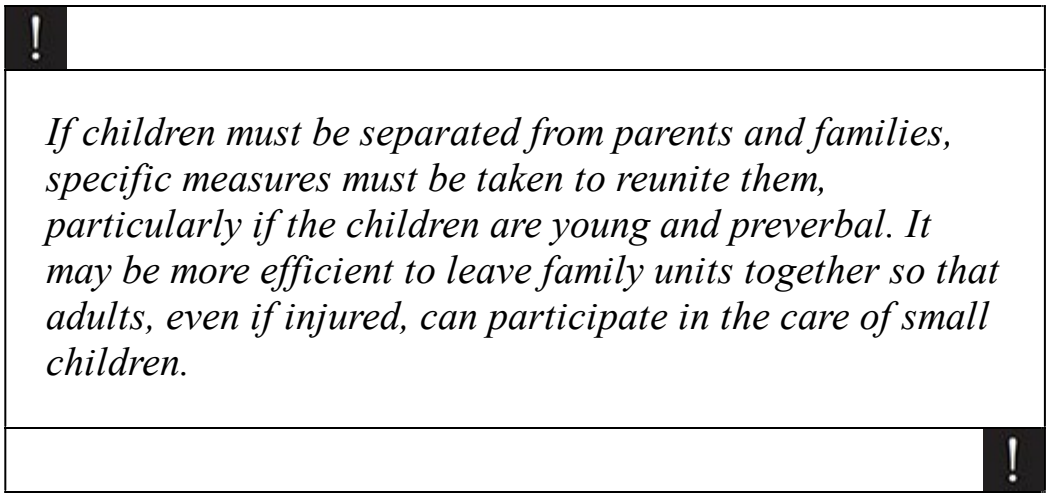
VI. PRESERVING THE FAMILY UNIT

Children depend on family and caregivers. Adults often place the needs of children above their own, and it may be difficult or inadvisable to separate children from adults in a disaster. If children must be separated from their parents and families, specific measures must be taken to reunite them, particularly if the children are young and preverbal. It may be more efficient to leave family units together so that adults (even injured adults) can participate in the care of small children. When family members are separated, children are at risk for intentional injury, exposure, and accidents in the postdisaster period. If family members must be separated, emergency workers must get complete identification, and personnel must be assigned to the task of caring for the child until the family can be reunited.

Attention must be given to caring for the entire family unit. After Hurricane Katrina, children who were separated from their parents during the evacuation remained separated afterward and suffered psychological trauma. When children are evacuated with adults, the relationships must be identified,

and guardianship status must be determined.

In a disaster that occurs without warning, children may be at school or day care. Disaster plans should not assume that children will be in the custody of their parents when disaster strikes. Planning must take into account children who are not injured but need sheltering and evacuation. Although that may not be the job of hospital-based healthcare providers, pediatricians must ensure that local and regional planning addresses the requirements of those who are not injured but cannot take care of themselves.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VII. ADAPTING DISASTER PLANS TO ADDRESS THE NEEDS OF CHILDREN

The needs of children are consistently overlooked in disaster planning in part because so many other shortcomings exist in the planning process. Preparations made for adults may apply to children when plans are tailored to include the pediatric population.

A. Emergency and Prehospital Planning

Pediatric-specific triage protocols should be taught to and practiced by first responders at the local, state, and national levels, and support for their development must come from the federal level. The specific educational needs of physicians, nurses, and respiratory care providers who will likely care for children in a disaster should be assessed. Emergency medical services personnel and response vehicles should be furnished with pediatric-specific equipment and medications. Pathways for communication, referral, and transport should be assessed and bolstered as necessary. The emergency medical services command center must recognize local hospital resources and capabilities in caring for children. Knowledge of the availability of services like pediatric surgery, trauma, and critical care is needed for directing ambulances to the appropriate facility. Information on bed occupancy further helps distribute patients and prevent overcrowding at any hospital.

B. Hospital Planning

Most children's hospitals take the lead in pediatric disaster planning, but in local incidents, frantic parents will choose the nearest emergency room. Thus, disaster planning must prepare for situations in which the number of children who require medical care is out of proportion to normal numbers. When children are affected in proportion to their share of the total population, the hospital census will show a higher than normal percentage of pediatric patients. If a disaster occurs at a school, at a day care center, or on a school bus, the number affected could be even greater. Children may need to be cared for in non-pediatric units.

In general, hospitals that do not routinely care for large numbers of children must include pediatricians on their disaster management planning teams. If pediatricians lack training in disaster management, appropriate education should be provided. The pediatricians, in turn, can identify and enhance areas of planning where the needs of children differ from those of adults. Plans to manage a surge of pediatric patients may include organizing teams in which care providers with pediatric-specific skills supervise those without such skills.

Pediatric disaster planning should include the 3 *S*'s: staff, supplies, and space.

1. Staff: A roster of all personnel who care for children in an emergency should be available, including physicians, nurses, respiratory therapists, pharmacists, child life specialists, and social workers. The most critical children are expected to be under the care of pediatric critical care physicians; however, neonatologists, adult emergency physicians with pediatric experience, anesthesiologists, and mid-level providers (eg, physician assistants and nurse practitioners) should be prepared to participate in the care of children in a disaster.

2. Supplies: The availability of both age- and weight-appropriate equipment is crucial, as is knowledge of the number of available pediatric beds, cribs, ventilators, oscillators, and other equipment.

3. Space: Designated locations for critically ill and injured pediatric patients must be identified; these include the pediatric emergency department, pediatric inpatient units, pediatric ICU, postanesthesia care unit, and adult, surgical, and medical ICUs, if necessary. The number of beds with monitoring capability should be noted. Surge plans for disasters should recognize that patients may not be able to be safely transferred to a tertiary center if roads and bridges are closed, and that hospital staff may face similar barriers in coming to work. Communication and modes of transport between community hospitals and children's hospitals should be detailed, including written transfer agreements that specify triage criteria, preferably with a predetermined regional incident command structure. Equipment and pharmaceutical supplies should be evaluated for appropriateness for children. In institutions that do not regularly care for children, supplies may need to be augmented with pediatric-specific equipment, pharmaceuticals, and supplies (eg, decontamination units and DuoDote auto-injector kits). Staff drills should include pediatric patients.

Disaster planning must include the needs of children in situations in which the number requiring medical care

is out of proportion to usual numbers.

When large numbers of families are affected, units that normally do not provide care for entire families may need to be adapted to this need. Plans for such changes be prepared and drilled in advance.

Children's hospitals must serve as referral centers that take the majority of pediatric casualties when feasible and should set the standards for pediatric disaster management.

Key Points

Pediatric Emergency Preparedness

- It is incumbent on caregivers and medical professionals to develop plans for disaster response well in advance of any incident.
- Community and local hospital plans should include pediatric disaster planning.
- Prehospital triage systems are applied during a disaster when the main goal becomes allocating limited resources quickly to salvageable patients.
- Simple interventions, such as supplemental oxygen and IV fluids, can save many injured children.
- Disaster responders must be familiar with the immature anatomy and developing physiology of the child and with vulnerabilities unique to the child.
- Disaster plans should include caregiving for uninjured children and family reunification.

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MANAGEMENT OF THE POISONED CHILD AND ADOLESCENT

Objectives

- Outline the strategies to resuscitate, evaluate, and stabilize the poisoned patient.
- Compare indications and contraindications of decontamination and elimination techniques.
- Review common toxidromes, their presentations, and therapies.
- Explore two specific poisonings and therapy.

Case Study

Yesterday, a 17-year-old boy with a history of depression and multiple suicide attempts was started on a new dose of the monoamine oxidase inhibitor, tranylcypromine. Today, he was found agitated, diaphoretic, and shaking; he admitted to taking dextromethorphan to "feel good." En route to the emergency department, he had a seizure. His vital signs upon presentation are: temperature, 41.2°C (106.1°F); heart rate, 195 beats/min; respiratory rate, 32 breaths/min; and oxygen saturation on room air is 95%.

Detection

– What is this patient's physiologic status?

– What should be included in the differential diagnoses?

Intervention

– What are the immediate treatment priorities?

– Does the airway need to be secured?

– Are any fluid boluses required for resuscitation?

Reassessment

– Is the treatment strategy effective?

– What is the etiology of the patient's altered mental status?

– Is any history available from the family?

– Is this teen at risk for ongoing toxin exposure or absorption?

– Is any decontamination required?

Effective Communication

– Does the poison center need to be consulted?

– When the patient's clinical status changes, who needs the information and how will it be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

I. INTRODUCTION

There are two major groups of poisoned children: those younger than 6 years, in whom 99% of poisonings are unintentional; and adolescents, in whom intentional causes must be considered. Children younger than 6 years tend to ingest a single agent in a small amount, which is usually nontoxic, and they present for medical attention shortly after the ingestion. Adolescent poisoning tends to result in more deaths because of suicide attempts, mixed ingestions, experimentation, intentional abuse, and delay in seeking medical attention. Most poison exposures in children are the result of acute ingestion.

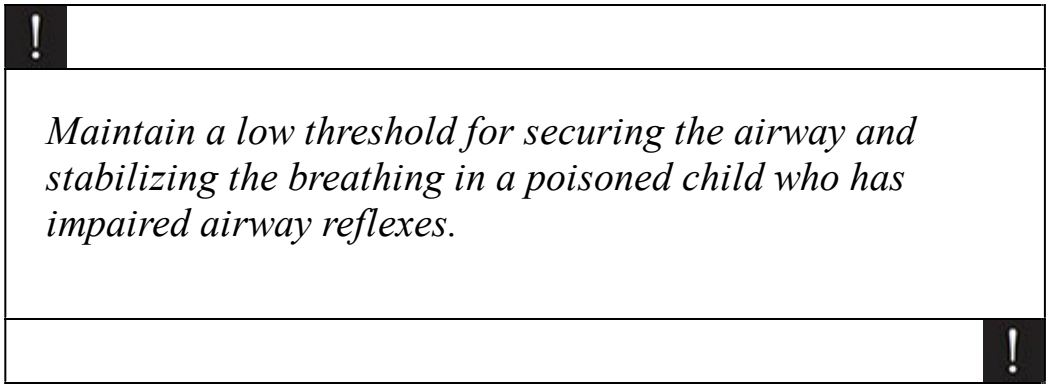
In all age groups, the most common class of substance exposure is analgesics, while the most common substances resulting in fatalities in developed countries are sedatives/hypnotics/antipsychotics. Other drug classes that result in fatalities include cardiovascular drugs, opioids, stimulants, and street drugs. Acetaminophen (paracetamol) and salicylates consistently remain in the top 10 most common causes of poisoning fatalities.

In developing areas, fuels such as paraffin and kerosene, pharmaceuticals, pesticides, antimalarials, metals (including iron and lead), plants, and envenomation remain major sources of poisoning. Most acute poisoning cases are unintentional and are caused by industrial toxins (such as organophosphates and hydrocarbon fuels) and envenomations from snake, arachnid, and insect bites. Acute and chronic poisoning can also follow the ingestion of fake, tainted, and "traditional" medications and foodstuffs, and the consumption of toxic plants by curious or hungry children. Chronic poisoning by water and food contaminated with heavy metals is also relatively common. Pediatric substance abuse is comparably rare in most developing countries, with certain exceptions (ie, street children and child soldiers).

Morbidity is a significant problem, but global data are not available and regional data are not comparable because of differing access to services and hospital admission criteria. In general, the mortality rate for children who are poisoned is low: 7.5% for all younger than 19 years and 1.4% for US children younger than 6 years; this despite the fact that children represented 64% of poisoning exposures in 2014.

A. Resuscitation and Stabilization

As with all acutely ill children, the first step is prompt recognition and intervention in life-threatening conditions. A primary survey or rapid cardiopulmonary assessment focuses on the *ABCD* approach (airway, breathing, circulation, depressed level of consciousness, disability, drugs, decontamination). Vital signs should be obtained immediately and age-related norms considered. Isotonic fluids such as normal saline should be the first-line therapy for hypotension before the institution of vasopressors. Considerations during the initial management of the patient with a depressed level of consciousness should include administration of dextrose, naloxone, and flumazenil.


!
Maintain a low threshold for securing the airway and stabilizing the breathing in a poisoned child who has impaired airway reflexes.
!

Following stabilization, a secondary survey includes a more focused history to determine substance, quantity, and time of ingestion. In addition to vital signs and general physical examination, factors such as mental status, agitated or depressed state, pupillary size, presence of nystagmus, and seizure often suggest a specific toxicologic syndrome or *toxidrome*. Hypothermia, hypotension, or bradycardia suggests toxic ingestion of narcotics, sedative/hypnotics, or antihypertensives. Hyperthermia, hypertension, or tachycardia suggests anticholinergics, sympathomimetics, cocaine, or amphetamines. Dilated pupils are consistent with amphetamines, cocaine, anticholinergics, antihistamines, and sympathomimetics, while constricted pupils suggest narcotics, cholinergics, organophosphates, and phencyclidine. Nystagmus can be present with toxic ingestions of alcohols, carbamazepine, carbon monoxide, phencyclidine, ketamine, phenytoin, or sedative/hypnotics, while seizures can be caused by a number of agents (**Table 13-1**).

Table 13-1 SIGNS AND SYMPTOMS ASSOCIATED WITH PEDIATRIC INGESTIONS

Symptom	Toxic Causes	Clinical Tip
Hypoxic child	Carbon monoxide, methemoglobinemia, cyanide	
Hypoxia and pulmonary edema	Cocaine, amphetamines, metal fumes, nitrogen dioxide, opioids, salicylates, smoke inhalation	
Wheezing child	β-blockers, irritant gases (chlorine), hydrocarbons, isocyanates, organophosphates, carbamates, smoke inhalation, foodborne	

	sulfites	
Increased osmolar gap[a]	Acetone, ethanol, ethyl ether, ethylene glycol, isopropyl alcohol, mannitol, methanol, propylene glycol, renal failure and ketoacidosis (diabetic and alcoholic)	Calculated osmolality = 2 x Na^+ (mEq/L) + glucose (mg/dL)/18 + blood urea nitrogen (mg/dL)/2.8. Normal osmolar gap (between measured and calculated) = 3-10 mOsm/kg
Increased anion gap[a]	Methanol, uremia, diabetic ketoacidosis, paraldehyde, iron, inhalants (carbon monoxide, cyanide, toluene), isoniazid, ibuprofen, lactic acidosis, ethylene glycol, ethanol, ketoacidosis, salicylates	Normal anion gap = $[Na^+]$ - ($[Cl^-]$ + $[HCO_3^-]$). Normal = 8-12 mEq/L
Methemoglobinemia	Antimalarials, benzocaine, dapsone, bipyridylium, inhaled nitric oxide, lidocaine, naphthalene, nitrates, nitrites, nitroethane, nitroprusside, phenazopyridine, prilocaine, sulfonamides	
Tachyarrhythmia	Amphetamines, cocaine, caffeine, chloral hydrate, aromatic hydrocarbons, anticholinergics, theophylline	
QT prolongation	Amiodarone, arsenic, chloroquine, quinine, quinidine, organophosphates, tricyclic antidepressants	More agents have been shown to prolong QT; check current references. If the cause of QT prolongation is sodium channel blockade, administration of 1-2 mEq/kg sodium bicarbonate or 3% saline may be helpful.
Sleepy child	Antihistamines, any sedative/hypnotic, alcohols, *γ*-hydroxybutyrate, tricyclic antidepressants, opioids, carbon monoxide, cyanide, hypoglycemia,	Naloxone or flumazenil may be diagnostic for reversing opioids, or benzodiazepines, respectively. Glucose hypoglycemic agents administration will reverse hypoglycemia.
Seizures	Amphetamines, anticholinergics, antihistamines, butyrophenones, caffeine, camphor, carbamates, carbon monoxide, cocaine, cyanide, ethylene glycol, hypoglycemic agents, methanol, 3,4-methylene- dioxymethamphetamine (MDMA), meperidine, isoniazid, lithium, nicotine, organophosphates, phencyclidine, phenothiazines, phenylpropanolamine, tricyclic antidepressants, salicylates, strychnine, theophylline, venlafaxine, *Gyromitra* (mushroom species)	Pyridoxine for isoniazid-induced seizures Avoid phenytoin, fosphenytoin and barbiturates with isoniazid ingestion.
Tachycardia	Amphetamines, caffeine, cocaine, theophylline, carbon monoxide, cyanide, hydrogen sulfide, anticholinergics (antihistamines, phenothiazines, tricyclic antidepressants, and atropine), ethanol, withdrawal from any psychoactive drug	
Bradycardia	Digoxin, organophosphates, carbamates, physostigmine, β-blockers, clonidine, opioids, calcium channel blockers, lithium	

[a]Please see **Chapter 8** for an expanded discussion regarding osmolar and anion gaps.
Classified using Pulsus Group, Inc. Koren G. A primer of paediatric toxic syndromes or 'toxidromes.' *Paediatr Child Health*. 2007;12:457-459.

With the possible exception of a point-of-care or serum blood glucose level or arterial blood gas measurement, laboratory results in poisoned patients are often not necessary for the initial resuscitation and stabilization. At the start of treatment, the determination of the anion and osmolar

gaps, electrolytes, renal function, and specific serum quantitative tests can provide valuable information in mixed or unknown ingestions, and can help assess the need for antidotes or elimination therapies. Quantitative tests in pediatric toxicology (**Table 13-2**) should always include acetaminophen/paracetamol and salicylate levels as they represent common, treatable, and potentially lethal ingestions. The results of qualitative tests, such as those obtained using a urine toxicology screen, provide confirmation, assist in evaluation of altered mental status, and help determine the need for counseling. Other supportive tests include an electrocardiogram, co-oximetry, venom detection kits, and pregnancy testing in girls of childbearing age.

Table 13-2	USEFUL QUANTITATIVE TESTS IN PEDIATRIC TOXICOLOGY

- Acetaminophen (paracetamol)
- Anticonvulsants (e.g., carbamazepine, phenytoin, valproic acid)
- Barbiturates
- Carboxyhemoglobin
- Digoxin
- Ethanol
- Ethylene glycol
- Iron
- Lead
- Lithium
- Methanol
- Methemoglobin
- Salicylate
- Theophylline

Adapted with permission. © 2005 McGraw-Hill. Hoffman RJ. Laboratory testing. In: Erickson TB, Ahrens WR, Aks SE, et al, eds. *Pediatric Toxicology*. 1st ed. New York, NY: McGraw-Hill; 2005:151-159.

B. Interventions

After basic stabilization is ensured, the use of specific interventions or therapies, including antidotes and enhanced elimination techniques, is limited to cases in which the expectation is that a defined benefit outweighs the risk of the procedure. Information is limited in distinguishing lethal concentrations of certain drugs and toxic substances in children. There is also limited pediatric experience with therapies such as lipid emulsion therapy. Therefore, indications for interventions in children may be different from those in adults, and their use needs to be carefully considered before implementation.

Decontamination is the next step, as it will limit further absorption of the toxin. Methods include gastrointestinal, environmental, and external decontamination.

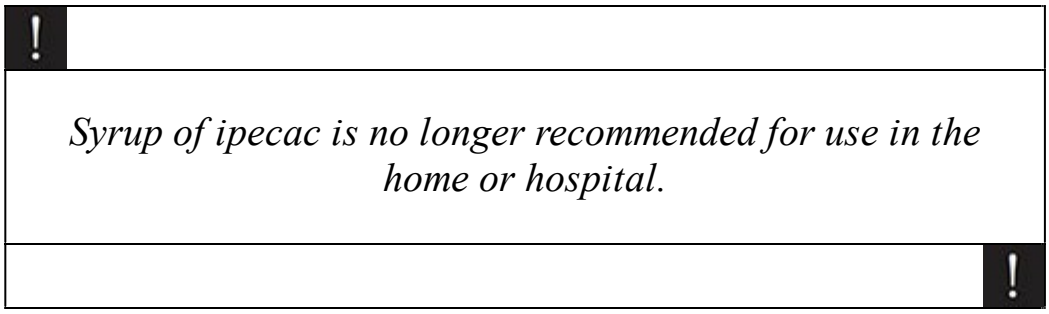
C. Gastrointestinal Decontamination

Gastrointestinal (GI) decontamination refers to measures to minimize absorption of the ingested toxin from the GI tract. Historically, many approaches have been adopted, including gastric evacuation (forced emesis or gastric lavage), intragastric binding (most commonly by single or multidose activated charcoal), and enhancing the transit of toxins to decrease total absorption time (whole bowel irrigation or cathartics). As clinical practice has evolved and an understanding of the efficacy, risks, and benefits of decontamination has grown, many practices have fallen out of favor. No controlled clinical studies have demonstrated that “routine” GI decontamination reduces morbidity and mortality in poisoned patients. However, evidence suggests that decontamination may reduce the absorption of toxins and may be helpful in select circumstances. The decision to perform GI

decontamination is based upon the specific poison(s) ingested, the time from ingestion to presentation, the presenting symptoms, and the predicted severity of the poisoning.

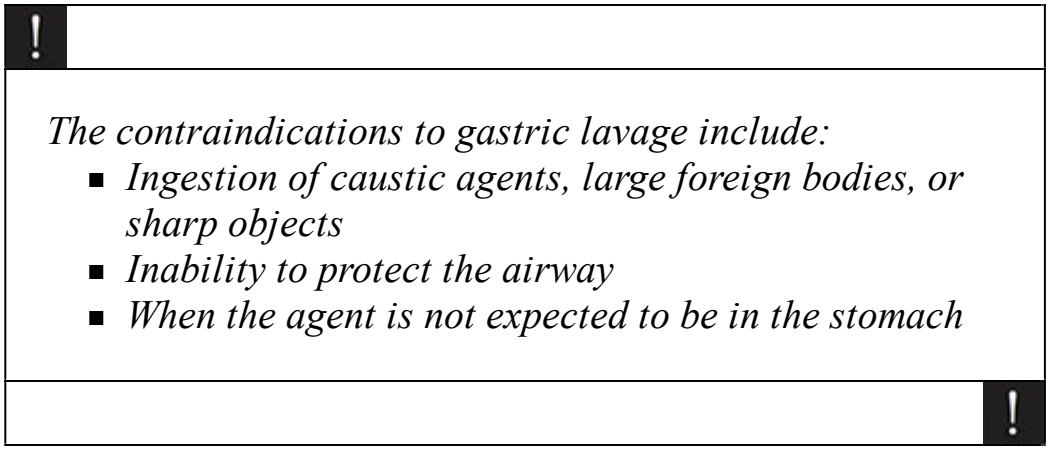
1. Syrup of Ipecac

Syrup of ipecac has not been shown to demonstrate any benefit, may interfere with charcoal administration, and may lead to complications like aspiration pneumonitis. As such, the American Academy of Pediatrics and the American Academy of Clinical Toxicology have recommended that ipecac no longer be used to treat ingestions.



2. Gastric Lavage

Gastric lavage, using a large orogastric tube and either saline or water, has no definite indications, with studies demonstrating lack of improvement in outcome. Thus, routine use is not recommended by the American Academy of Clinical Toxicology. Lavage may be considered in patients with known life-threatening overdoses; it is only performed when a benefit is anticipated and 1 of the following conditions exists: the substance does not bind to activated charcoal; the opportunity to use activated charcoal is significantly delayed or missed; the child presents within 1 hour of ingestion without significant central nervous system (CNS) symptoms, or when even a minimal decrease in toxic exposure might be critical, such as with ingestion of calcium channel blockers or lithium. The contraindications to gastric lavage include: ingestions of caustic agents, large foreign bodies, and sharp objects; the inability to protect the airway; and when the agent is not expected to be in the stomach. Complications include aspiration, esophageal or gastric perforation, decreased oxygenation during the procedure, hyponatremia, and water intoxication. The presence of a cuffed endotracheal tube may decrease, but may not eliminate, the risk of aspiration.


!
The contraindications to gastric lavage include:
Ingestion of caustic agents, large foreign bodies, or sharp objects
Inability to protect the airway
When the agent is not expected to be in the stomach
!

3. Activated Charcoal

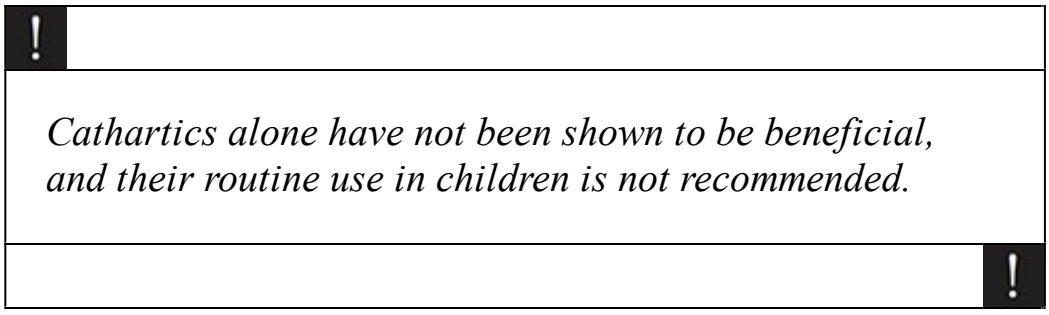
Activated charcoal (AC) has an increased particle surface area to adsorb the toxins from the GI tract. It is most likely to be of benefit if administered for identified serious poison exposures with early presentation (<1 hour after ingestion) and before the onset of symptoms, especially CNS symptoms. However, patients with ingestions that delay gastric motility may benefit from later administration because of the slowed absorption and gastric emptying. Additionally, delayed AC administration may be beneficial in salicylate ingestions. The recommended dose is 1 g/kg, with a maximum recommended dose of 50 g. AC is effective for most tablet ingestions, although not for such agents as iron, lithium, alcohols, strong acids and alkalis, cyanide, many pesticides, and hydrocarbons; however, when these substances are ingested, AC may still be indicated because of co-ingestants. In the setting of life-threatening ingestions, such as carbamazepine, dapsone, phenobarbital, salicylates, phenytoin, quinine, and theophylline, multiple doses of AC have been shown to be useful in lowering toxic blood levels and shortening the course of substances that undergo enterohepatic or enterogastric recirculation. However, the use of multiple doses has not been demonstrated to improve patient outcomes. Aspiration is a potential complication, so AC should not be administered unless the patient is able to protect the airway or the trachea has been intubated. AC is contraindicated in patients with suspected bowel obstruction or perforation.

4. Whole Bowel Irrigation

Whole bowel irrigation (WBI) using a polyethylene glycol solution (given orally or by nasogastric tube) produces a rapid catharsis with expulsion of the GI contents, but there is no convincing evidence from clinical studies that it improves outcomes. WBI may be considered in potentially toxic ingestions of sustained-release or enteric-coated medications and drugs that are not well adsorbed by charcoal, such as iron, lithium, and lead. WBI may also facilitate removal of ingested packets of illicit drugs in “body packers.” Small children should receive 0.5 L/h, whereas older children and adolescents should receive 1.5-2 L/h until rectal effluent is clear, which typically occurs after an infusion for 4 to 6 hours. The irrigation solutions can liberate toxins from AC and potentially interfere with its efficacy; therefore, AC should be administered before WBI. Contraindications to the use of WBI include the presence of ileus, bowel obstruction, bowel perforation, hemodynamic instability, and unprotected airway.

5. Cathartics

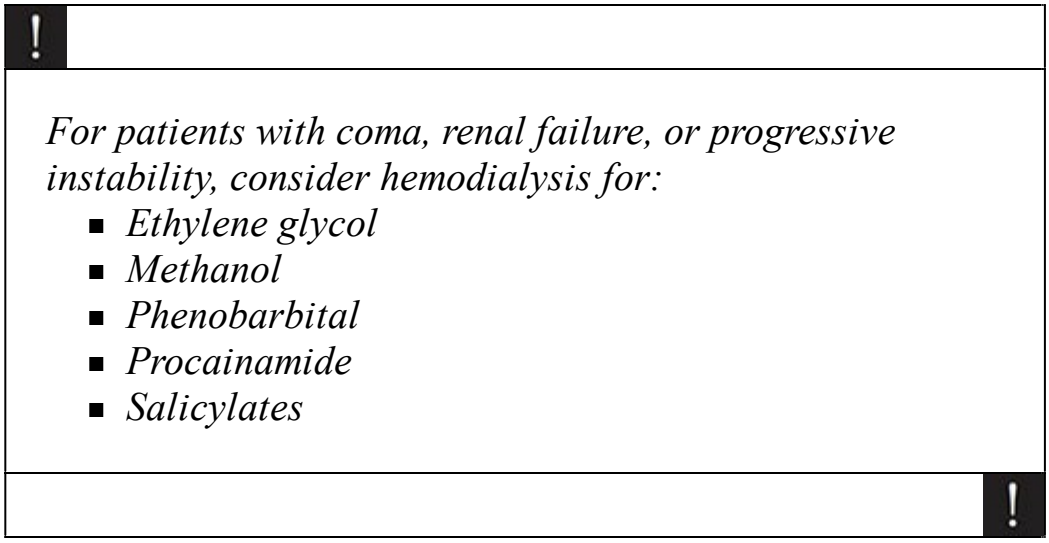
Cathartic agents, alone or in combination, have not been shown to be beneficial, and their routine use in children is not recommended. However, because salicylate levels may increase due to its release from AC, administration of AC mixed in sorbitol is reasonable. Sorbitol has shorter transit times than any magnesium preparation.



D. Enhanced Elimination

Forced diuresis, urinary alkalinization, hemodialysis, continuous renal replacement therapies, and charcoal hemoperfusion are attempts at enhanced toxin elimination. They have varying efficacies and depend on a substance's volume of distribution and usual route of elimination. Forced diuresis has been shown to be ineffective and is not recommended. Alkaline diuresis can increase the elimination of weak acids, such as salicylates and phenobarbital, and is indicated with ingestion of these agents. A general goal of urinary alkalinization via the administration of IV sodium bicarbonate is to maintain a urine pH >7.5.

Extracorporeal measures to enhance the elimination of toxins may be useful in certain dangerous poisonings with substances that can be retrieved from the vascular compartment. Hemodialysis uses a semipermeable membrane to remove specific toxins from the blood against a concentration gradient. It should be used for substances that have small molecular weights, are water soluble, have a small volume of distribution, and are not highly protein bound. Patients who are poisoned with substances causing renal failure, coma, or progressive instability, and who meet the appropriate pharmacokinetic criteria, should be considered as candidates for hemodialysis. These substances include ethylene glycol, methanol, phenobarbital, procainamide, lithium, valproic acid, carbamazepine, and salicylates.



Charcoal hemoperfusion uses a cartridge of AC to remove toxins from the blood and can be used for overdoses involving carbamazepine, phenobarbital, procainamide, and theophylline.

E. Lipid Emulsion Therapy

Lipid emulsion therapy (LET) is increasingly considered as a rescue antidote to treat lipophilic drug toxicities, such as anesthetics, calcium channel blockers, and tricyclic antidepressants. This therapy has a number of known complications, including lipemia interfering with laboratory testing, pancreatitis and associated symptoms of abdominal pain, nausea and/or vomiting, and acute respiratory distress syndrome. Because of limited data associated with lipid emulsion therapy, its use is reserved for hemodynamically unstable patients in whom supportive efforts are not successful.

F. External Decontamination

Children must be removed from contaminated air if it is the source of the poisoning. Ocular contamination requires copious irrigation. In the case of dermal exposure to toxins, any residual agent must be removed as soon as possible, with care taken to protect others from exposure, including the healthcare providers. Running large volumes of lukewarm soapy water over the naked child is safe for most toxins, but not certain powder-based chemical weapons. Attention must be paid to the child's core temperature to avoid hypothermia. Contaminated clothing and runoff can be a source of secondary intoxication. Some volatile agents are toxic when exhaled, as are certain poisons in the vomitus and feces.

II. TOXIDROMES

The most common toxidromes seen in children are summarized in **Table 13-3**. Recreational drugs resulting in hospital admissions are listed in **Table 13-4**.

Table 13-3 COMMON TOXIDROMES

	Signs and Symptoms										
Toxidromes	**BP**	**Pulse**	**RR**	**Temp**	**Mental Status**	**Pupil size**	**Peris-talsis**	**Diaphor-esis**	**Reflexes**	**Other**	**Common Causative Agents**
Anticholinergic	N ↑	↑	±	↑	Delirium	↑	↓	↓	N	Dry mucous membranes, flush, urinary retention, mumbling speech, picking movements; mnemonic: "blind as a bat, mad as a hatter, red as a beet, hot as a hare, and dry as a bone"	Antihistamines, atropine, antipsychotic drugs, scopolamine, tricyclic antidepressants
Cholinergic (muscarinic)	±	↓	N ↑	N ↓	Normal to depressed	N ↓	↑	↑	N	Mnemonic SLUDGE: salivation, lacrimation, urination, diarrhea, GI upset and emesis; bronchorrhea	Organophosphates, physostigmine, pilocarpine, pyridostigmine
Cholinergic (nicotinic)	↑	↑	±	N	Depressed	↑	±	N ↑	↓	Fasciculations, paresis, abdominal pain, headache	Nicotine

Opioid	N ↓	N ↓	↓↓	N ↓	Depressed	↓	↓	N	N ↓	Some opioids (meperidine, tramadol) may not cause miosis	Opioids, heroin
Sedative/ hypnotic	N ↓	N ↓	N ↓	N ↓	Depressed	N ↓		↑	N ↓	Ataxia	Barbiturates, benzodiazepines (clonazepam, alprazolam)
Sympatho-mimetic	↑	↑	↑	↑	Agitated	↑	N ↑	↑	↑	Tremor, seizures	Amphetamines, cocaine, caffeine, cathinone, phencyclidine, theophylline
Serotonin syndrome	↑	↑	N ↑	N ↑	Variable	↑	N ↑	↑	↑	Clonus and rigidity greater in lower extremities	Citalopram, fluoxetine, paroxetine, sertraline
Withdrawal (ethanol/ sedative hypnotic)	↑	↑↑	↑	↑	Agitated, disoriented	↑	↑	↑	↑	Tremors, seizures	Ethanol, barbiturates, benzodiazepines
Withdrawal (opioids)	↑	↑	N	N	Normal, anxious	↑	↑	↑	N	Vomiting, rhinorrhea, piloerection, diarrhea, yawning	Opioids

BP, blood pressure; RR, respiratory rate, Temp, temperature; ↑, increased; ↑↑, very increased; N ↑, no change or increased; ±, variable; N, change unlikely; ↓, decreased; N ↓, no change or decreased; GI, gastrointestinal

Table 13-4 COMMON CAUSES OF RECREATIONAL POISONING

Agent/Club Drug[a]	Mechanism of Toxicity	Clinical Features	Management
Amphetamine (eg, methamphetamine, paramethoxyamphetamine) *Speed, meth, ice, uppers, death*	Sympathomimetic: adrenergic receptor stimulation	**Cardiovascular:** tachycardia, hypertension, palpitations, arrhythmias, coronary ischemia **Metabolic:** hyperthermia, rhabdomyolysis, diaphoresis **Nervous system:** seizures, intracranial hemorrhage	• Supportive care and monitoring • Fluid and electrolyte balance • Benzodiazepines for seizures, hypertension, and sedation • Active cooling measures – ice packs; may need sedation and paralysis • Sodium bicarbonate for wide complex tachyarrhythmias
Ecstasy (3,4-methylene-	Sympathomimetic,	See amphetamines + hyponatremia, seizures,	• Avoid β-blockers

dioxmethamphetamine [MDMA]) *XTC, E, eccies, adam*	serotonergic, SIADH	bruxism	and phenytoin
Cocaine *Snow, crack, coke, rock*	Sympathomimetic, local anesthetic	See amphetamines action, cardiac Na channel blockade	• Antihypertensive agents • Nitrates and opiate analgesia for coronary ischemia • Sodium bicarbonate for wide complex tachyarrhythmias
Ketamine (phencyclidine is similar) *Special K, Vitamin K, ket, cat valium*	Sympathomimetic	See amphetamines + altered sensory perception, injuries and trauma, emergence phenomena	• Consider multisubstance abuse
Dextromethorphan *DM, Dex*	Pro-serotonergic	Dissociative "high," dysphoria; if taken with MAOI or SSRI, will present with serotonin syndrome	• Patients may have anticholinergic appearance if the combination product contains antihistamine. • Check acetaminophen levels.
Nitrates (amyl nitrate, butyl nitrate) *Blue bottles, poppers, liquid incense*	Vasodilation, heme moiety oxidation	Hypotension, syncope, methemoglobinemia	• Supportive care, methylene blue for methemoglobinemia
γ-Hydroxybutyrate (and precursors, GBL, BD) *GHB, GBH-grievous bodily harm, easy lay, liquid G*	GABA receptor activation	Sedation, coma, myoclonus, seizures	• Supportive care
Benzodiazepines *Valium, Xanax, Ativan*	GABA receptor activation	Sedation, coma, hypotension	• Supportive care, flumazenil
Anticholinergic agents *Angel's trumpet,* Datura *sp*	Muscarinic receptor blockade	Delirium, dry skin and membranes (inability to sweat), flushing of skin, hyperthermia, visual impairment (mydriasis, loss of lens accommodation), urinary retention ("blind as a bat, red as a beet, hot as a hare, dry as a bone, mad as a hatter")	• Low stimulus, benzodiazepine sedation, physostigmine
Methylene-dioxypyrovalerone (MDPV), mephedrone *Bath Salts, Ivory Wave, Vanilla Sky*	Sympathetic stimulation	See amphetamines + profoundly altered mental status, sweating, mydriasis, muscle tremors and spasms, stroke, cerebral edema, respiratory distress, myocardial infarction, severe panic attacks, agitation, paranoia, hallucinations, and violent behavior	• Supportive care • Benzodiazepines (for sedation, to control seizures, or both) • IV fluids, especially if suspicion of rhabdomyolysis

Dihydrodesoxy-morphine-D, desomorphine *Crocodile*	8-15 times more potent than morphine, typically has impurities	Similar to morphine + skin in places of injection becomes grey and green, scabrous, flakes off so it resembles the skin of a crocodile; may cause peripheral limb ischemia with necrosis and possible secondary infection; may smell of iodine	• Supportive care • Naloxone
Synthetic cannabinoid receptor agonists *K2, spice*	Similar to cannabis intoxication, possibility of violent behavior, persistent psychosis, geno-toxicity	Agitation, anxiety, paranoia, tachycardia, hypertension, muscle spasms, seizures, dystonia, diaphoresis, tremors, psychosis	• Aggressive supportive care; • Benzodiazepines for agitation and seizures • EEG for seizures or unexplained AMS • Serial cardiac enzymes for chest pain

[a] Street names are given in italics (representative sample).
SIADH, syndrome of inappropriate antidiuretic hormone secretion; MAOI, monoamine oxidase inhibitor; SSRI, selective serotonin reuptake inhibitor; GBL, gamma-butyrolactone; BD, 1-2-butanediol; GABA, gammaaminobutyric acid; AMS, altered mental status; EEG, electroencephalogram.

A. Sympathomimetic

Sympathomimetic poisoning syndrome presents with hypertension, tachycardia, hyperthermia, seizures, CNS excitation, and mydriasis. Cocaine and amphetamines, including methamphetamine, 3,4-methylenedioxymethamphetamine (MDMA, Ecstasy), and cathinone ("bath salts"), are the typical ingestants. Additional findings on physical examination include tremor, warm skin, diaphoresis, hypoactive bowel sounds, restlessness, paranoia, hallucinations, mania, and insomnia. Treatment is mainly supportive with benzodiazepines and short-acting antihypertensive agents for easy titration when necessary. Patients should be monitored for myocardial ischemia, stroke, cardiac arrhythmia, and rhabdomyolysis.

B. Narcotic

Overdose with a narcotic agent presents with a triad of miosis, respiratory depression, and depressed level of consciousness. Ingestion of heroin, morphine, or codeine can present with these findings, as well as hypotension, bradycardia, hypothermia, hypoactive bowel sounds, hyporeflexia, and confusion. Treatment for opioid toxicity is supportive, with naloxone as a specific antidote. The half-life of naloxone may be much shorter than that of the opioid, and repeat doses – or rarely, an infusion – might be required. Also, the administration of naloxone might result in unmasking of pain, tachycardia, and systemic and pulmonary hypertension; thus, it should be used with caution in patients at risk of pulmonary hypertension. Administration to a patient with chronic exposure to narcotics may precipitate a severe withdrawal crisis.

C. Sedative/Hypnotic

Sedative or hypnotic poisoning presents with a depressed level of consciousness, respiratory depression, bradycardia, hypotension, and hyporeflexia. Typical agents include benzodiazepines and, less commonly, barbiturates. Additional findings could include confusion, delirium, ataxia, blurry vision due either to miosis or mydriasis, nystagmus, and hypothermia. The mainstay of therapy is observation and supportive care, with cautious consideration of flumazenil as it may induce seizures. Once again, the half-life of flumazenil is relatively short and repeated doses might be needed if apnea recurs. Flumazenil is not recommended in the patient who received benzodiazepines to control seizures with resultant apnea/respiratory depression, and it is contraindicated in comatose patients if the diagnosis has not yet been made. Its use in tricyclic antidepressant overdose and in those regularly taking benzodiazepines may precipitate intractable seizures.

D. Anticholinergic

The anticholinergic toxidrome includes thirst, hyperthermia, mydriasis, delirium, tachycardia, tachypnea, and dry skin, the latter of which differentiates anticholinergic and sympathomimetic toxidromes. Causative agents include atropine, diphenhydramine, antihistamines, tricyclic antidepressants, and antipsychotics. Many hallucinogenic plants (ie, *Datura*, angel's trumpet, jimson weed) are anticholinergics. Other presentations could include hypertension, psychosis, chorea, seizures, coma, depression, confusion, hallucination, and urinary retention. Treatment is supportive with a focus on cooling and antihypertensive therapy. Benzodiazepines can be helpful in the agitated patient.

E. Cholinergic

The cholinergic syndrome is classically described by the mnemonic *SLUDGE*: salivation, lacrimation, urination, defecation, GI upset, emesis, and the "killer B's," namely bronchorrhea, bronchospasm, and bradycardia. Organophosphates, found in some insecticides and in nerve gases like sarin, are agents that inhibit cholinesterase so that acetylcholine accumulates in the affected tissues. SLUDGE represents the muscarinic receptor toxicity, in addition to bradycardia, tachypnea, hypothermia, miosis, and bronchorrhea, while nicotinic receptor stimulation produces tachycardia, hypertension, fasciculations, abdominal pain, confusion, weakness, paralysis, drowsiness, coma, and mydriasis. Children may present only with the central symptoms and with tachycardia instead of bradycardia, as nicotinic effects predominate over the muscarinic effects; however, miosis is usually present, as well as a garlic odor, which may suggest the diagnosis. Atropine, a competitive inhibitor of only the muscarinic acetylcholine receptor, and pralidoxime, an enzyme that reverses cholinesterase inhibition at both muscarinic and nicotinic receptors, are effective antidotes.

III. SPECIFIC AGENTS AND ANTIDOTES

Common toxins and their antidotes are listed in **Table 13-5**; however, a more detailed discussion of acetaminophen/paracetamol and salicylate poisonings is warranted as they represent frequent, potentially fatal, but treatable ingestions.

Table 13-5	TOXIC INGESTIONS AND SPECIFIC THERAPIES

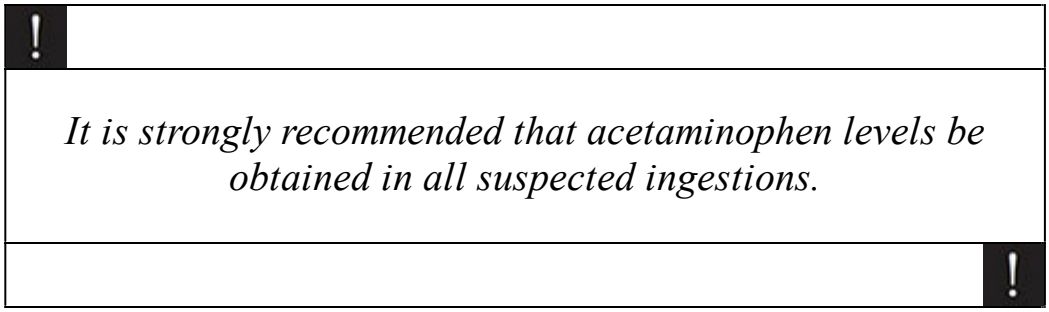
Agent	Therapy
Acetaminophen	*N*-acetylcysteine
Amphetamine	Benzodiazepine
Anticholinergics	Physostigmine
Anticholinesterase	Atropine, pralidoxime
Anticoagulants	Vitamin K_1, protamine
Benzodiazepine	Flumazenil*
ß-adrenergic blocker	Glucagon
Botulism	Botulinum antitoxin, BIG (botulism immune globulin)
Calcium channel blocker	Calcium, glucagon, insulin
Carbamate	Atropine, pralidoxime
Carbon monoxide	Oxygen, hyperbaric oxygen
Cholinergic	Atropine, pralidoxime
Cocaine	Benzodiazepine
Cyanide	Amyl and sodium nitrite, thiosulfate, hydroxocobalamin
Digoxin	Digoxin-specific Fab fragments
Ethylene glycol	Fomepizole, ethanol
Heavy metal	British antilewisite, ethylenediaminetetraacetic acid (EDTA), penicillamine, 2,3-dimercaptosuccinic acid (DMSA)
Heparin	Protamine
Hypoglycemic agent	Glucose, octreotide, glucagon
Iron	Deferoxamine
Isoniazid	Pyridoxine
Lead	British antilewisite, 2,3-dimercaptosuccinic acid (DMSA)
Lithium	Hemodialysis
Methanol	Fomepizole, ethanol
Methemoglobin	Methylene blue
Nitrites	Methylene blue
Opioids	Naloxone, nalmefene
Organophosphate	Atropine, pralidoxime
Phenothiazine	Diphenhydramine
Salicylate	Sodium bicarbonate, hemodialysis
Sulfonylureas	Dextrose, octreotide

Theophylline	Charcoal, hemoperfusion
Tricyclic antidepressant	Sodium bicarbonate
Warfarin derivative	Vitamin K_1, fresh frozen plasma

* Flumazenil may precipitate seizures when used in patients who are chronic benzodiazepine users or in situations of tricyclic antidepressant overdose.

A. Acetaminophen/Paracetamol

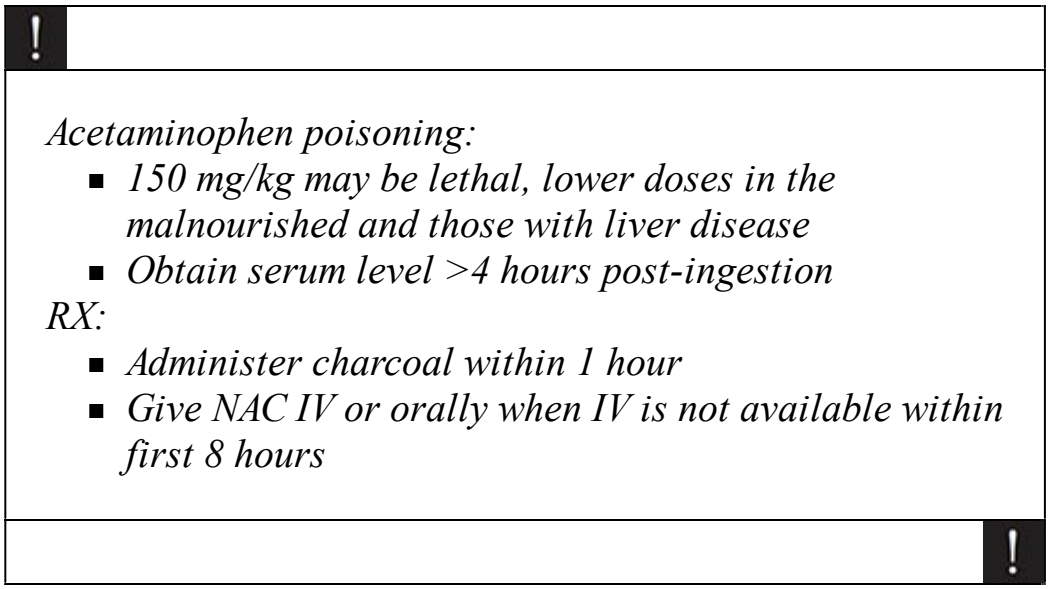
Acetaminophen is one of the most commonly reported potentially toxic pharmaceutical agents and the cause of the greatest absolute number of fatalities from poisoning in developed countries. Acetaminophen is contained within hundreds of prescription and nonprescription analgesics, both alone and in combination; it is a prevalent co-ingestant and has an increased incidence of therapeutic overuse. With the recognition of its role as a frequent cause of hepatotoxicity, liver failure, and death, and the availability of an effective antidote, it is strongly recommended that acetaminophen levels be obtained in all suspected ingestions.



Metabolism in the liver is predominantly by glucuronidation and sulfonation; when these systems are overwhelmed by toxic ingestions, the cytochrome P450 system produces a highly reactive and toxic metabolite, N-acetyl-para-benzoquinone imine (NAPQI). This is usually reduced by glutathione into a nontoxic metabolite, but when glutathione stores are depleted, NAPQI increases and causes hepatocellular damage. Less lethal toxicity is seen in children as they have more glutathione than adults.

An acetaminophen dose of 150 mg/kg is potentially toxic, although much lower doses can be fatal in children with glutathione depletion (ie, malnutrition) and chronic liver disease. Drug levels should be obtained 4 hours after ingestion, or as soon as possible thereafter, to assess the risk of toxicity using the Rumack-Matthew nomogram. The nomogram is not useful earlier than 4 hours post-ingestion as distribution following a single acute ingestion will not have occurred. A second level should be obtained 4 hours after the first when extended release formulations are suspected. The nomogram is only useful for acute exposure and does not apply to patients on long-term acetaminophen therapy. The antidote for acetaminophen poisoning is *N*-acetylcysteine (NAC), which acts as a precursor for glutathione and increases nontoxic sulfation metabolism, among other mechanisms. NAC, available in both oral and intravenous forms, is most effective if administered within 8 hours of ingestion, but it should be given whenever acetaminophen toxicity is suspected. The entire course of therapy must be administered to every patient with an acute ingestion and a toxic acetaminophen level, even if the plasma acetaminophen level becomes negative during the treatment, as NAC is being used to treat

effects of the toxic metabolite, not the parent compound. Activated charcoal binds to NAC, but losses due to charcoal binding are not clinically significant; therefore, charcoal should not be withheld in isolated acetaminophen ingestion presenting within 4 hours or with a potential co-ingestant.



Acetaminophen poisoning:

- *150 mg/kg may be lethal, lower doses in the malnourished and those with liver disease*
- *Obtain serum level >4 hours post-ingestion*

RX:

- *Administer charcoal within 1 hour*
- *Give NAC IV or orally when IV is not available within first 8 hours*

B. Salicylates

The overall incidence of salicylate poisoning has declined, but still 15 US fatalities occurred in 2014. These agents have the potential to be a problem as they can be found in a number of over-the-counter oral and topical preparations, such as bismuth subsalicylate and oil of wintergreen. Physiologically salicylate poisoning initially causes a respiratory alkalosis by direct stimulation of the medulla, followed by an increased anion gap metabolic acidosis through a number of mechanisms. Thus, the initial blood gas measurement may be misleading to the uninitiated. Patients present with nausea, vomiting, tinnitus, cerebral edema, CNS disturbances, hyperthermia, coagulopathy, pulmonary edema, tachypnea, tachycardia, hepatotoxicity, rhabdomyolysis, hypoglycemia, hypokalemia, and dehydration. Infants and children may not manifest the initial respiratory alkalosis.

Ingestion of >150 mg/kg of salicylate should be considered toxic. A ferric chloride test—where 1 to 2 drops of $FeCl^3$ in 1 mL of urine produces a purple color change in the presence of any salicylate, or the Phenistix test (Miles Laboratories), which produces a brown color change— may be performed as a screen when salicylate levels are not readily available. A quantitative test will be necessary to determine therapy. In acute ingestions, symptoms are typically present with levels >30 mg/dL (2.15 mmol/L). Treatment objectives include cardiopulmonary stabilization, prevention of absorption, correction of fluid deficits, correction of acid-base abnormalities, and enhancement of excretion and elimination. The treatment plan should include GI decontamination with gastric emptying in an acute overdose and activated charcoal for large ingestions. Aggressive fluid resuscitation is an important first step. To achieve a urine pH between 7.5 and 8, urine alkalinization to trap ions and thereby prevent tubular absorption in the kidney should be instituted for serum levels >40 mg/dL (2.2 mmol/L) or in the presence of signs and symptoms of salicylism. Acetazolamide is not recommended; although it alkalinizes the urine, it acidifies the serum.

Severe poisoning is signaled by salicylate blood levels >100 mg/dL (7.14 mmol/L) in acute ingestions or 40 mg/dL in chronic ingestions, significant neurotoxicity (agitation, coma, convulsions),

kidney failure, pulmonary edema, or cardiovascular instability. Patients with acute ingestion whose serum levels are >100 mg/dL (7.14 mmol/L) should undergo dialysis. In chronic ingestions, serum levels are unreliable in predicting the severity of illness; however, a level >60 mg/dL (4.3 mmol/L) may be a relative indication for dialysis, as are seizures, altered mental status, refractory acidosis, or deterioration despite adequate supportive care.

Salicylate poisoning:

- *>150 mg/kg is toxic*
- *Symptoms with blood levels >30 mg/dL*

RX:

- *Use GI emptying and activated charcoal*
- *Alkalinize urine for serum level >40 mg/dL*
- *Consider dialysis for serum levels in acute ingestion > 100mg/dL, chronic ingestion >60 mg/dL*

C. Antivenin

Antivenins are available for certain reptile and arthropod envenomations. All venoms are complex mixtures; any bite resulting in symptoms indicates a poisoning with the potential for serious multisystem effects. Children are at greater risk than adults because of their small body size relative to the amount of venom injected. Most antivenins are purified polyvalent immunoglobulins produced by injection of small amounts of venom into dedicated horses or sheep. As foreign proteins, antivenins have a risk of inducing hypersensitivity reactions, both type I (immunoglobulin E-mediated hypersensitivity) and type III (immune complex-mediated hypersensitivity). In view of this and their cost, they are indicated only for cases of envenomation, not after all bites. High-quality antivenins exist for all major venomous Australian, European, and North American snakes; many Asian, African, and South American snakes; various arthropods; and the Australian box jellyfish (*Chironex fleckeri*) and stonefish. It is important to ensure that the product used is appropriate for the likely species, has been appropriately stored, and is a genuine product from a reputable supplier. Low-quality and fake antivenins are a major problem in several countries, particularly in Asia and Africa.

Key Points

Management of the Poisoned Child and Adolescent

- Following the *ABCD* of resuscitation, administration of dextrose, naloxone, or flumazenil should be considered in patients who present with a depressed level of consciousness.

- Neither flumazenil nor naloxone should be administered in any patient with a history of chronic benzodiazepine or narcotic use, respectively, as administration may precipitate drug withdrawal.
- Initial blood tests should always include acetaminophen/paracetamol and salicylate levels, as these agents represent common treatable and potentially lethal ingestions.
- Syrup of ipecac is no longer recommended for use.
- The decision to perform GI decontamination is based upon the specific poison(s) ingested, the time from ingestion to presentation, the presenting symptoms, and the predicted severity of the poisoning.
- Knowledge of the symptoms of common toxidromes will allow rapid identification of the most likely cause and timely initiation of appropriate treatment of the poisoned patient.

Suggested Readings

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TRANSPORT OF THE CRITICALLY ILL CHILD

Objectives

- Describe the key elements for both safe intrahsopital and interhsopital pediatric transport.
- Outline the communication process for the safe transport of the acute and critically ill child.
- Specify the information the referring institution needs to provide to the transport team and accepting facility to facilitate stabilization, appropriate referral, and timely transfer.
- Outline methods for stabilization of the acute and critically ill child prior to transfer.
- Identify the factors that influence transport team composition and the mode of transport.
- Anticipate and recognize serious problems and complications related to transport.
- Describe the role and opportunities of telemedicine and other advanced technologies in patient transport.
- Itemize and identify the equipment and monitoring necessary for safe transport and optimal outcomes.

Case Study

A 4-year-old boy who is intubated secondary to respiratory distress and hemodynamic compromise is being transported to a regional children's hospital. The child is being transported with infusions of intravenous fluids, sedation and vasoactive medications. During the transport, the child becomes hypotensive, with worsening perfusion.

Detection

– What is this child's physiologic status?

– What are the most likely diagnosis and the worst possible diagnoses?

Intervention

– What are the priorities to stabilize the patient?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– What information should be communicated to the medical control person?

– What type of equipment should be accompanying this patient on transport?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

I. INTRODUCTION

Critically ill pediatric patients are at increased risk of morbidity and mortality during transport to access care that is not available at the existing location, such as care in a diagnostic department, operating room, specialized unit within the hospital, or another medical facility. The decision to transport these patients is based on an assessment of the potential benefits versus the potential risks associated with the transport itself. Risk is not isolated to the patient but may extend to some degree to the accompanying personnel. The risks of transport can be minimized by careful planning, the use of appropriately qualified personnel, the availability of appropriate equipment, and optimal timing.

Risk to the patient during transport can be minimized through careful planning, appropriate transport modality, use of appropriately qualified personnel, and the selection and preparation of appropriate equipment.

II. GENERAL CONSIDERATIONS

Initially most critically ill or injured children are taken to the closest medical facility for primary stabilization. Once stabilized, many of these children will require transport to a facility with an appropriate level of care and expertise. Interhospital transport services are a vital link in the system of emergency and critical care for children, bridging the patient's initial stabilization with definitive care in the tertiary care center. This transport from one hospital to another should be from a lower

level of care to a higher level of care.

During transport, a patient is potentially at risk for instability and deterioration. Therefore, transport for a diagnostic test or a procedure that is unlikely to alter the management or outcome must be questioned. Whenever possible, diagnostic testing or simple procedures should be attempted at the bedside to avoid subjecting a patient to the inherent risks associated with transport.

The transport team is selected according to training and skills, and the available equipment and supplies are assembled to provide for any ongoing or anticipated needs for acute care. There should be minimal disturbance in the monitoring or maintenance of the patient's vital functions during transport. Ideally, all transports of critically ill pediatric patients, both intrahospital and interhospital, should be performed by specially trained individuals. However, in rural or austere environments, this is not always possible and local resources may determine team composition and mode of transport.

Given that not all transferring facilities have access to a dedicated pediatric critical care transport team, contingency plans for using locally available resources should be developed and frequently reviewed. The standards for organized transport services as set forth by the American Academy of Pediatrics (AAP) and the Society of Critical Care Medicine (SCCM) are rigorous. Other professional organizations such as AMPA (Air Medical Physician Association), ASTNA (Air and Surface Transport Nurse Association) and IAFP (International Association of Flight Paramedics) continue to set the standard and provide resources for Air and Ground Transport teams. Since 1999 the Commission of Accreditation of Medical Transport Systems has been the main accrediting body for the transport industry in the United States. This entity provides guidelines for all aspects of transport. 183 transport teams maintain accreditation across the United States as of February 2017. ASTNA and IAFP provide training and guidance for transport team configuration, safety measures, and general standards. They also provide advance certification opportunities for Registered Nurses and Paramedics practicing as critical care transport teams.

A comprehensive and effective interhospital transfer plan for facilities that do not have access to an organized pediatric critical care transport team can be developed in 4 steps:

1. A multidisciplinary team of physicians, nurses, respiratory therapists, hospital administrators, and local emergency medical services personnel is formed to plan and coordinate the process.
2. The team conducts a needs assessment that focuses on patient demographics, transfer volume, transfer patterns, available resources (personnel, equipment, emergency medical services, communication), and receiving facilities.
3. A standardized transfer plan is written including a medical control protocol to cover the care of the patient in transit for unexpected emergencies or in case of deterioration.
4. The transfer plan is regularly evaluated and refined after use and at periodic intervals using a standard quality improvement process.

Transport to a tertiary care setting should be coordinated with the receiving providers to ensure that the patient travels safely and arrives in stable or improved condition. Ideally, the transport is supervised in person or in communication with a physician who has experience and training in pediatric emergency medicine or pediatric critical care.

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The mode of transport and the composition of the transport team should be based on the acuity and complexity of care required by the individual patient.

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The mode of transportation for a critically ill patient is an important aspect of comprehensive care. Ground ambulances, helicopters, and fixed-wing aircraft are the principal vehicles available for interfacility transport. Depending upon geographic location and resources, other vehicles may be needed, including, but not limited to, boats, private vehicles, carts, and bicycles. The composition of the accompanying team will also depend upon the resources available to the geographic location and should include individuals with the skills and experience that best meet the needs of the patient during transport.

Pediatric transport medicine is a relatively new field of clinical interest that has developed rapidly over the past three decades. The AAP has stated that the goal of pediatric interfacility transport is to improve outcomes of critically ill or injured pediatric patients who are not in proximity to a hospital that provides the required level of care. Advances in technology and transportation have allowed dedicated pediatric critical care transport teams to offer many therapies that were previously restricted to use within the intensive care unit (ICU). These types of specialized teams can minimize transport-associated risks and improve outcomes.

Every pediatric tertiary care facility should have an organized pediatric transport system. Ideally, the system is regional, with central medical control under the direction of a physician trained in pediatric emergency medicine or pediatric critical care medicine.

III. INTRAFACILITY TRANSPORT

The basic reason for moving a critically ill pediatric patient within the facility is the need for additional care that utilizes technology and/or specialists not available in the child's current location. This may require moving a patient within the hospital to a diagnostic testing area, operating room, or pediatric intensive care unit (PICU). The transport process must be organized and efficient due to the potential risks it entails for the patient. Intrahospital transport is not benign and physiological adverse events can occur during transfer. Safe transport of the critically ill pediatric patient requires coordination, communication, and appropriate equipment and monitoring to ensure stability and prevent clinical deterioration in the patient's clinical condition.

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The key elements of intra- and interfacility transport are planning, communication, personnel, equipment, and mode

of transportation.

A. Pre-transport Communication and Coordination for Intrafacility Transport

Physician-to-physician and nurse-to-nurse communication regarding the patient's condition and treatment should occur both prior to and at the completion of any transport (**Appendix 13**). Detailed handoff communication should occur whenever a patient's management will be assumed by a different team, whether related to transfer from unit to unit, diagnostic testing, or interventions performed outside the PICU.

Communication between transport team members and the accepting clinical team occurs each time responsibility for patient care is transferred from one location to another and is achieved through the use of detailed hand-offs of clinical care provided to the patient during transport.

Pre-transport confirmation should indicate that the area to which the patient is being transported (eg, radiology, operating room, nuclear medicine) is ready to receive and immediately begin the specified procedure or test. Ancillary services and other members of the healthcare team (eg, security, respiratory therapy, escort) should be notified of the timing of the transport and the equipment and monitoring that will be required. The responsible physician should either accompany the patient or be notified that the patient is out of the PICU and at risk of an acute adverse event in another area of the hospital. Monitoring and resuscitation equipment must be equivalent to that of the PICU and, if necessary, additional monitoring should be provided during transport to ensure safe transport. The medical record should document the indications for transport and the patient's status and interventions performed during the transport process. Maintaining communication with the responsible physician and the transport team during procedures which take place in a diagnostic or radiology suite is critical in providing safe care for patients during intrafacility transport.

B. Personnel During Intrafacility Transport

It is strongly recommended that a minimum of 2 people accompany a critically ill pediatric patient. One should be a nurse who has completed a competency-based orientation and has met the prescribed standards for pediatric critical care nurses to include being a Pediatric Advanced Life Support Provider (American Heart Association (AHA), 2015). Additional personnel may include a respiratory therapist, registered nurse, or critical care technician as needed. It is strongly advised that a healthcare professional (physician, nurse practitioner, physician assistant) with training in airway

management, advanced pediatric life support, critical care, or the equivalent accompany intubated or unstable critically ill patients.

When a procedure is anticipated to be lengthy and the receiving location is staffed by appropriately trained personnel, patient care may be transferred to those individuals if this action is acceptable to both groups. This allows for maximum utilization of staff and resources. If care responsibility is not transferred, the transport personnel should remain with the patient until return to the PICU or monitored acute care areas eg. emergency room.

C. Equipment and Monitoring During Intrafacility Transport

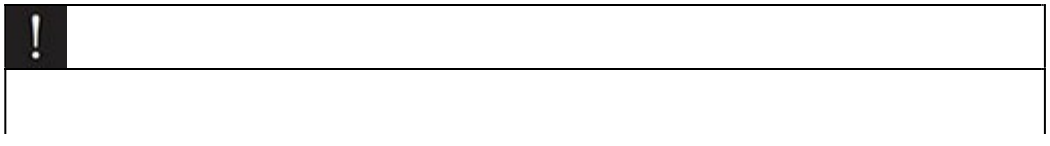
All critically ill patients require a blood pressure monitor, a pulse oximeter, and a cardiac monitor/defibrillator. At the very minimum, the transported patient should receive continuous electrocardiography monitoring, continuous pulse oximetry, and periodic measurement of blood pressure, pulse rate, and respiratory rate. Invasively ventilated patients should have end-tidal carbon dioxide monitoring, and this is also advisable for those undergoing procedural sedation or who have other risk factors for respiratory depression. It is recommended that transport monitoring be equivalent to the level delivered in the ICU. Continuous intra-arterial blood pressure monitoring, central venous pressure and intracranial pressure monitoring may be beneficial in select critically ill patients.

When available, monitors with the capacity for storing and reproducing patient bedside data should be utilized so that data collected during the procedure and transport can be reviewed. The minimum requirements are shown in **Table 14-1**.

Table 14-1	MINIMUM EQUIPMENT REQUIRED FOR TRANSPORT

- Electrocardiographic monitor
- Pulse oximeter
- End-tidal CO_2 capable monitor
- Defibrillator with battery backup and transcutaneous pacing capability
- Oxygen tank with sufficient supply for duration of transport, plus 30 minutes' reserve
- Ventilator appropriate for infants, children, and adults
- Resuscitation bag with appropriately sized masks
- Infusion pumps with battery backup
- Noninvasive blood pressure monitor
- Portable suction unit
- Standard resuscitation drugs

Airway management equipment, including a resuscitation bag and face mask of proper size and fit for the patient's age and size, should accompany the patient. It is also important to have an oxygen source of ample supply to provide for the patient's projected needs during transport plus an additional 30 minutes of oxygen in reserve.



When a critically ill child requires transport, the same level of monitoring that occurs in the intensive care unit or acute care unit should be maintained throughout the transport.

Standard resuscitation drugs, such as epinephrine (adrenaline) and antiarrhythmic agents, should be transported with each patient in the event of sudden cardiac arrhythmia or arrest. A more complete array of pharmacologic agents should either accompany the patient or be available from supplies in the emergency resuscitation carts or crash carts located along the transport route and at the transport destination. In many hospitals, pediatric patients share diagnostic and procedural facilities with adult patients, so a complete set of pediatric resuscitation equipment and medications must accompany infants and children and also be available at their destination. Supplemental medications, such as narcotics and sedative agents, are to be considered on a case-by-case basis.

An appropriate supply of intravenous fluids and continuous drip medications, administered and regulated on battery-operated infusion pumps, should be ensured. All battery-operated equipment must be fully charged and capable of functioning for the duration of the transport. Backup batteries and electric cords for the equipment should be included. If a healthcare professional capable of medical decision making will not be accompanying the patient during transport, protocols or communication mechanisms must be in place to permit the administration of medications and fluids by appropriately trained personnel under emergency circumstances.

Hand ventilation with manual resuscitation bag is the most commonly employed ventilation method during intrahospital transports. However, portable mechanical ventilators are gaining in popularity and use because they are capable of more reliably administering prescribed minute ventilation and desired oxygen concentrations. In children, a default oxygen concentration of 100% is frequently used. However, oxygen concentration must be precisely regulated for neonates and patients with congenital heart disease who have single ventricle physiology or who are dependent on a right-to-left shunt to maintain systemic blood flow. For patients receiving mechanical ventilatory support, equipment similar to that used in the PICU is preferred at the receiving location; otherwise, the equipment used in the PICU is transported to meet the patient at the destination.

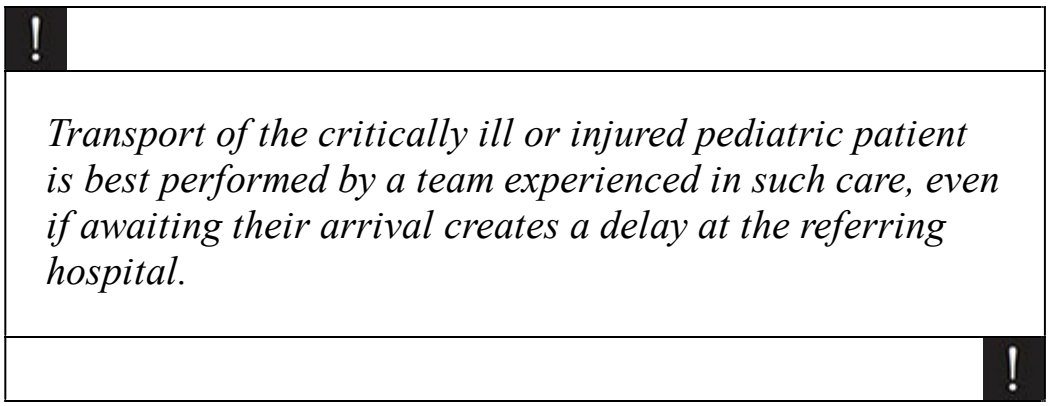
The position of a patient's endotracheal tube must be noted and should be confirmed radiographically before transport. The adequacy of oxygenation and ventilation is confirmed with either manual resuscitation bag ventilation or trial on transport ventilator. Occasionally, patients may require modes of ventilation or ventilator settings that are not replicable during transport or at the receiving location. In such cases, alternative modes of mechanical ventilation must be tested before transport to ensure appropriateness of therapy and patient stability. If a patient cannot be maintained safely with alternative therapy, the risks and benefits of the transport must be reexamined.

Transport ventilators must have alarms for both disconnection and excessively high airway pressures. Pediatric patients with tracheostomy tubes must be transported with great caution because most home or transport ventilators are not capable of sensing disconnection and alarms will be delayed. Any ventilator must have a backup battery power supply, and electrical cords must accompany the patient

during transport.

IV. INTERFACILITY TRANSPORT

The two general indications that a critically ill child requires interfacility transport are that the referring hospital lacks the resources to care for the patient beyond initial stabilization and/or that the patient has a diagnosis or severity of illness that requires comprehensive subspecialty care best accomplished at a regional pediatric tertiary care center. Transport should be arranged promptly when the referring facility has assessed the child and determined that its resources are not adequate or that subspecialty support is required.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Transport of the critically ill or injured pediatric patient is best performed by a team experienced in such care, even if awaiting their arrival creates a delay at the referring hospital.
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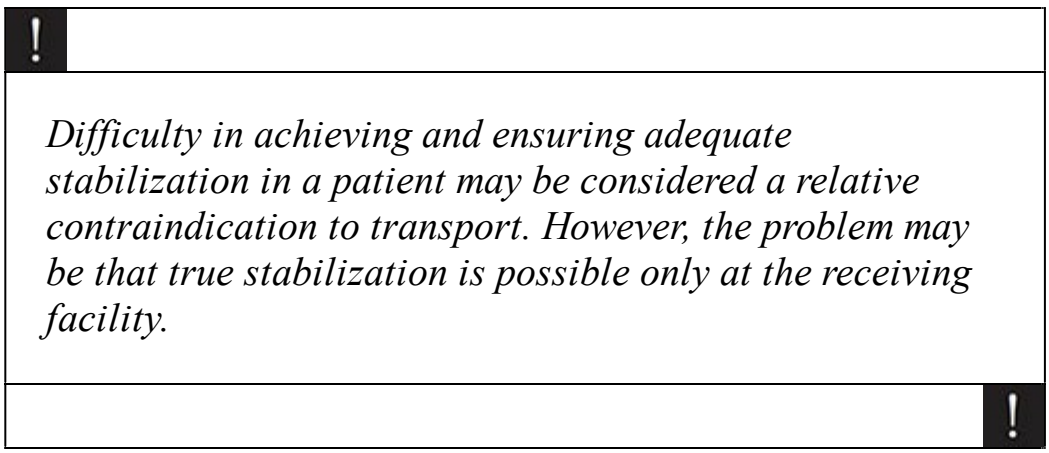
Timely recognition of a child in need of transport is critical. Favorable patient outcomes depend on the expertise and availability of nursing and medical personnel and the technologic support in the referral facility.

Delays are inherent in arranging an interfacility transport, particularly when a specialized team from the receiving hospital is necessary. Therefore, the child's clinical deterioration must be anticipated and expected, and an appropriate team must be mobilized as soon as possible during the initial stabilization. Firm open lines of communication between the referring hospital, the receiving hospital, and the transport team should be established and maintained throughout the process so that advice may be provided in an ongoing manner. Video links allow those not on-site to actually see the child and witness the treatment.


!
Difficulty in achieving and ensuring adequate stabilization in a patient may be considered a relative contraindication to transport. However, the problem may be that true stabilization is possible only at the receiving facility.
!

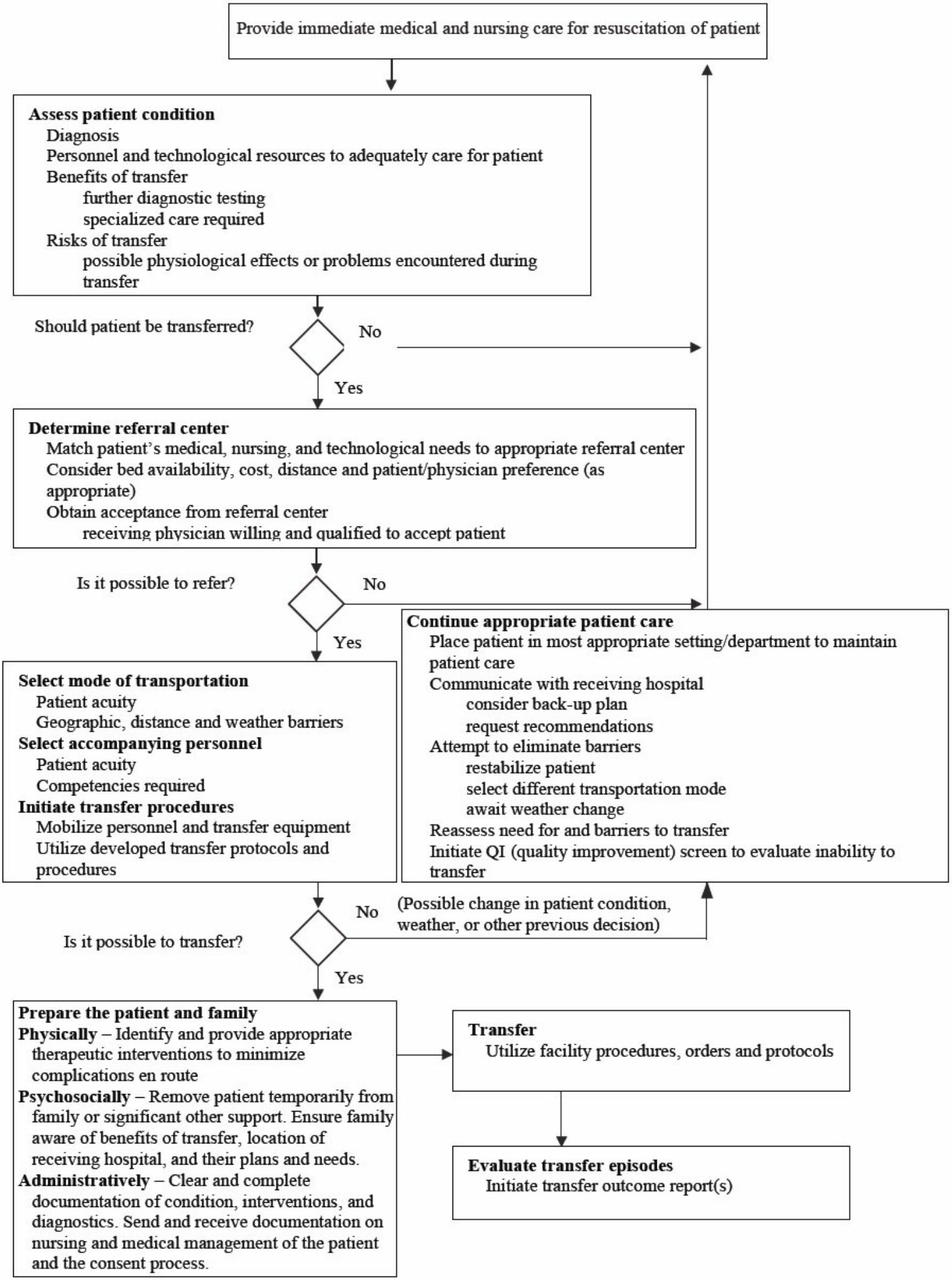
Rapid transport seldom is the sole concern in interfacility transport and rarely supersedes

stabilization. The level of care that can be provided during transport is an equally important consideration in recognizing and preventing clinical deterioration and providing continued stabilization. The transport team should be capable of providing an equal or higher level of care than the referring hospital and have the means to maintain that level of care during transport. The vehicle type, mode of transport, and team composition should be selected to best meet the needs of the patient during transport (**Appendix 13**).

Healthcare practitioners must be aware of their local, regional, and national laws regarding interfacility patient transfers. In the United States, federal regulations such as EMTALA (The Emergency Medical Treatment and Labor Act) define the legal responsibilities of the hospitals involved. Financially motivated transfers are illegal and put both the institution and the individual practitioner at risk for serious penalty. US regulations and best practices require that a competent patient, or the legally authorized representative of a minor child or incompetent patient, give informed consent prior to interfacility transport. This must include the disclosure of risks versus benefits of transport, documentation in the medical record, and a signed consent form. If circumstances do not allow for informed consent, both the indications for transport and the reason for not obtaining consent must be documented. Transfer and transport agreements must be completed in advance to prevent needless delays. AMPA has developed a volunteer web based databank for transport services to enter data. This initiative, known as GAMUT, hopes to allow for benchmarking of over 20 transport initiatives across the United States.

A transfer algorithm showing the sequence of events involved in an interfacility patient transport is presented in **Figure 14-1**.

Figure 14-1. Transport Algorithm


Provide immediate medical and nursing care for resuscitation of patient
Assess patient condition
Diagnosis
Personnel and technological resources to adequately care for patient
Benefits of transfer
further diagnostic testing
specialized care required
Risks of transfer
possible physiological effects or problems encountered during transfer
Should patient be transferred?
No
Yes
Determine referral center
Match patient's medical, nursing, and technological needs to appropriate referral center
Consider bed availability, cost, distance and patient/physician preference (as appropriate)
Obtain acceptance from referral center
receiving physician willing and qualified to accept patient
Is it possible to refer?
No
Yes
Continue appropriate patient care
Place patient in most appropriate setting/department to maintain patient care
Communicate with receiving hospital
consider back-up plan
request recommendations
Attempt to eliminate barriers
restabilize patient
select different transportation mode
await weather change
Reassess need for and barriers to transfer
Initiate QI (quality improvement) screen to evaluate inability to transfer
Select mode of transportation
Patient acuity
Geographic, distance and weather barriers
Select accompanying personnel
Patient acuity
Competencies required
Initiate transfer procedures
Mobilize personnel and transfer equipment
Utilize developed transfer protocols and procedures
No
(Possible change in patient condition, weather, or other previous decision)
Is it possible to transfer?
Yes
Prepare the patient and family
Physically – Identify and provide appropriate therapeutic interventions to minimize complications en route
Psychosocially – Remove patient temporarily from family or significant other support. Ensure family aware of benefits of transfer, location of receiving hospital, and their plans and needs.
Administratively – Clear and complete documentation of condition, interventions, and diagnostics. Send and receive documentation on nursing and medical management of the patient and the consent process.
Transfer
Utilize facility procedures, orders and protocols
Evaluate transfer episodes
Initiate transfer outcome report(s)

A. The Transport Process

Once the transport decision has been made, the patient must be prepared. The goal is optimal stabilization. The patient's condition and the trajectory of illness or injury must be considered. Whenever possible, medical interventions aimed at stabilizing the patient should be discussed with the receiving physician and the transport team. Airway patency must be assured.

> *If there is any concern about maintaining airway patency for the duration of the transport process, the patient must be intubated prior to transport.*

If necessary, intubation should be performed by the most experienced individual available, whether it is someone at the referring facility or a member of the transport team. Once the airway is stabilized and secured, adequate ventilation and oxygenation are ensured. Circulation must be assessed and stabilized. Maintaining adequate intravenous access is important. In most circumstances, two functional intravenous lines are preferred during transport. Appropriate temperature control is also essential, especially between buildings and within vehicles.

While the patient is being stabilized, the medical record, laboratory reports, radiographs, and any additional imaging studies are copied to accompany the patient. At least one parent or legal guardian should remain with the child during preparations, ensuring that necessary information and consent are obtained prior to the actual transport. Often, at least one parent or legal guardian may accompany the child throughout the transport process.

B. Mode of Transport

To ensure a successful interfacility transport, the most appropriate vehicle type and the best team composition should be requested. Interfacility transport is typically accomplished through the use of a ground ambulance, critical care mobile ICU ambulance, helicopter, or fixed-wing aircraft, but other modes of transport may be dictated by regional or local resources (e.g. private vehicle, bicycle, boat). The following factors must be considered when selecting the optimal mode of interfacility transport for a critically ill pediatric patient:

- Diagnosis and severity of illness
- Risk of deterioration during transport
- Team capabilities
- Urgency to arrive at the tertiary care facility and team response time

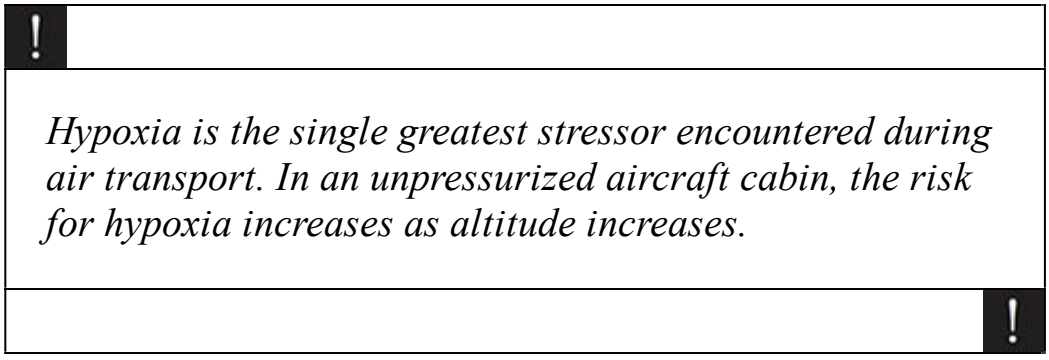
- Distance between the two facilities
- Local geography, weather, and traffic conditions
- Availability of vehicles or aircraft

After these factors have been weighed, the relative cost and availability of resources, both personnel and equipment, must be considered for each practical option.

The transport team must review the constraints of the transport environment, which is small and confined, with finite resources. Consequently, team members' sensory perception, communication, and range of motion are limited.

Compared to other modes of transport, ground ambulances are readily available, relatively inexpensive, and spacious. On average, they are the most common mode of interfacility transport. They are operable in most weather conditions and can be brought to a halt if interventions or procedures are required. In some cases, weather may necessitate that a ground ambulance be used, even if the distance between facilities is significant. Ambulances are generally considered safer than either helicopters or fixed-wing aircraft. Disadvantages associated with ground transportation include increased transport time over long distances and the risk of traffic and weather-associated delays.

The distinct advantages of a helicopter versus ground ambulance are faster transport to the receiving facility over a wider distance ranging from 100-150 miles (160-241 km), and avoidance of traffic congestion.Helicopters are best utilized when speed of transport is the most critical factor in the management of a patient. Monitoring and evaluating the pediatric patient are extremely difficult during helicopter transport. The small space inside the cabin and the constant noise and vibration from the rotors limit the ability to perform procedures during flight. In addition, helicopter transport is more costly than ground transport. Weather conditions preclude flying up to 15% of the time, limiting this mode of travel.


!
Hypoxia is the single greatest stressor encountered during air transport. In an unpressurized aircraft cabin, the risk for hypoxia increases as altitude increases.
!

Fixed-wing aircraft are typically used for transports of more than 150 miles (240 km). Depending upon the type of aircraft, the cabin environment may permit excellent patient monitoring and assessment. Many of these aircraft are pressurized and land at controlled sites. Interventions can be performed more easily than in rotor-wing aircraft. The disadvantages of fixed-wing transport are long start-up times (though this may be offset by the speed of the flight) and the need to move the patient multiple times, typically using a ground ambulance between the hospital and the aircraft. Multiple transfers increase the risk of such difficulties as airway deterioration, intravenous catheter malfunction, and equipment failure. Altitude may also increase the risk of adverse effects: hypoxia,

gas expansion, and reactions to temperature, humidity, and gravitational forces that are unique to air transport.

A patient's initial compensatory response to hypoxia is an increase in respiratory rate and effort. Supplemental oxygen should be provided, especially when a child's pulmonary status may be compromised. Additionally, as altitude increases, gas volume increases proportionally. Physiologically, a patient with a small pneumothorax may develop a clinically significant tension pneumothorax with increases in altitude due to decreases in barometric pressure. Similarly, pneumocephalus may expand to further complicate a closed head injury. Nasogastric tubes must be connected to a functioning suction unit to decompress the stomach. Pressurized equipment may need adjustment at high altitudes to maintain the appropriate negative pressure. The high-volume, low-pressure cuff on an endotracheal tube should be slightly deflated before flight, and periodically checked and adjusted during flight, to the minimal volume needed to maintain an adequate seal. Nonetheless, despite the many added considerations, the time saved by flying may overset the potential risks.

C. Configuration of the Transport Team

The primary responsibility for a patient typically stays with the referring institution until arrival at the accepting facility, unless the transport team is sent from the accepting facility. During transport, a patient should not be put at increased risk by any decrease in the level of care. Careful attention must be paid to the qualifications and experience of the transport personnel.

Variations in team composition will be dictated by the patient's needs and the hospital's resources. The composition will be dictated by the patient's needs and the hospital's available resources. The team may include physicians, nurses, respiratory therapists, paramedics, and emergency medical technicians. No single team composition is preferred over all others.

Team members may be obtained from local emergency medical services personnel, medical personnel from the referring hospital, hospital-based critical care teams that typically transport adult and trauma patients, and dedicated pediatric and neonatal transport teams. Patient safety and the availability of needed medical care are factors to consider when creating a team for a specific transport. Although local emergency medical systems tend to offer rapid response times, their personnel usually do not have the training, experience, or equipment to transport a critically ill or injured child. Healthcare professionals from the referring hospital may be rapidly mobilized, but their absence may place their facility at a disadvantage. Personnel with limited experience in pediatric prehospital or critical care will find patient management difficult in the mobile transport environment. Adult-based critical care transport services offer both ground and flight resources but often have limited expertise and equipment for the care of critically ill children.

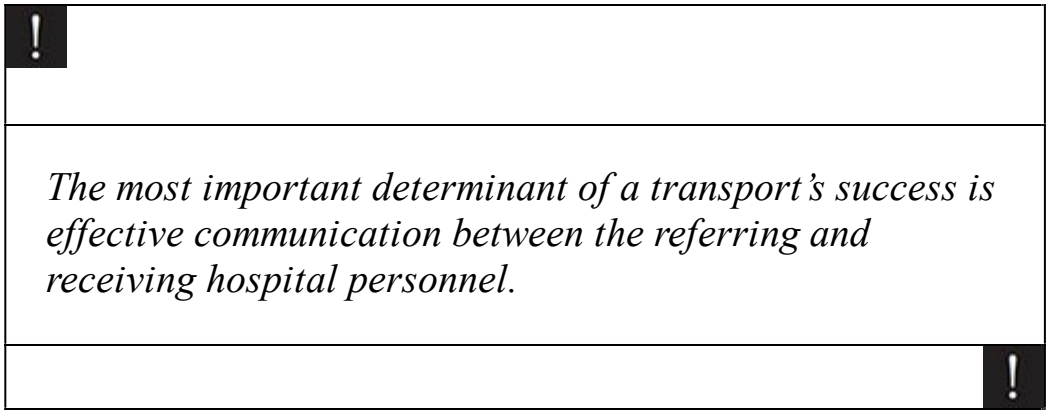
Specialized pediatric and neonatal transport systems offer personnel (nurses, respiratory therapists, nurse practitioners, physicians, and emergency medical technicians) who are skilled in the care of critically ill pediatric patients. These specialized teams offer the optimal level of care during transport and often provide continuity of care from transport to the PICU. They are usually based at the regional tertiary care center, making them somewhat slower to arrive at the referring facility, and they may be costly in comparison to other ground transport methods. Unfortunately, such teams are not

available in all areas, and they may not have access to all types of transport vehicles. When available, these teams should be used to transport the most unstable patients, even if they require more time to arrive or to stabilize a patient.

V. PREPARING A PATIENT FOR INTERFACILITY TRANSPORT

A. Pre-transport Coordination and Communication for Interfacility Transport

Every facility should establish a plan with protocols that outline the process for obtaining appropriate transport once it is determined to be needed. A list of names and telephone numbers of facilities equipped to manage critically ill children should be compiled and be readily available in the emergency department. There should also be a list of contact numbers for transport systems capable of accommodating these patients, if transport will not be provided by the receiving tertiary care facility.


!
The most important determinant of a transport's success is effective communication between the referring and receiving hospital personnel.
!

One of the most important steps in the interfacility transport of a critically ill pediatric patient is the initial referral call. Based on the information exchanged, the receiving hospital must determine the most appropriate bed assignment (PICU or general pediatric floor) and anticipate the patient's need for specialized diagnostic or therapeutic modalities. If a specialized pediatric transport team from the receiving hospital is mobilized, the optimal team composition must be determined. The key patient information for the initial referral call should include:

- Patient's identifying information
- Referring physician's name
- Facility name, location, and contact telephone number
- Vital signs and weight
- Brief summary of the patient's condition
- Pertinent findings of physical exam
- Relevant laboratory and radiographic results

- Significant interventions performed and patient response

The receiving physician may provide advice about further evaluation or interventions. Before transport can be initiated, that physician must accept the patient and confirm the availability of appropriate resources at the receiving hospital. The mode of transport will be determined by the transferring physician in consultation with the receiving physician and based on patient acuity, time, weather, any medical interventions required for continued stabilization, and available personnel and resources.

If the transport team is not associated with the referring or receiving facility, the staff at the referring hospital must assist in the arrangements for efficient patient transfer. The transport provider should be contacted to confirm availability, capabilities, and details of the patient's status and anticipated needs during transport. An estimate of preparation and transport time should also be shared (**Table 14-2**).

Table 14-2	INFORMATION REQUIRED BY THE TRANSPORT TEAM

- Patient's name, weight, age, sex, date of birth
- Patient's location (unit and hospital facility) at both transferring and receiving hospitals, including addresses and telephone numbers
- Names and contact telephone numbers for both transferring and receiving attending physicians
- Basic diagnosis
- Method of transport (ground, fixed-wing aircraft, helicopter)
- Nature of transport (incubator, pediatric stretcher, adult stretcher)
- Team composition (number and discipline(s) of team members)
- Reason for mode of transport (patient acuity, traffic, distance or weather conditions, etc.)
- Equipment required or anticipated for transport
- Patient status, including interventions already instituted

Classified using Freedman SH, King BR. Aeromedical transport procedures. In: Henretig FM, King C, eds. *Textbook of Pediatric Emergency Procedures*. Baltimore, MD: William & Wilkins; 1997.

A nurse-to-nurse report should be provided to the unit at the accepting facility. If that receiving facility is not involved in the transport process, the referring physician should telephone the receiving physician immediately before the patient's departure and report the most recent vital signs, current clinical status, and estimated time of arrival.

While awaiting the arrival or coordination of the transport team, staff at the referring hospital should ensure that the patient is optimally prepared for transport. The medical record and laboratory and radiographic studies should be copied, monitoring and assessment should be continued, and necessary procedures should be performed by the most experienced personnel. If airway patency or ventilatory status is questionable, the airway should be secured with an endotracheal tube and position confirmed radiographically. All intravascular lines should be checked for patency and taped securely in place. The cervical spine and any fractured bones should be stabilized and/or immobilized before transport. The patient should be given nothing by mouth throughout the transport process, and anti-emetics should be considered in those with an unprotected airway. Nasogastric tubes should be connected to a functioning suction unit. If clinically significant pneumothorax or hemothorax is present, a chest tube must be inserted and fluid or air collections evacuated prior to transport. In addition, the patient's family members should be informed of the nature of the child's illness or injury and why transport is

being arranged so that they can provide informed consent. The responsibilities of the referring hospital are summarized in **Table 14-3**.

Table 14-3	RESPONSIBILITIES OF THE REFERRING HOSPITAL

- Stabilize patient.
- Communicate with accepting facility early in the process.
- Notify the family and obtain consent for transfer/transport.
- Secure vascular access and tracheal tube.
- Stabilize the cervical spine and any fractures (as indicated).
- Copy all patient records and radiographic studies, including transfer summary, medication list, and laboratory results.
- Provide transport team with contact details (ie, telephone number, email, website) for results of pending laboratory studies.
- Prepare blood products, if indicated.

B. Personnel for Interfacility Transport

In addition to the vehicle operators, a minimum of two people should accompany the patient. At least one person must be a nurse, physician, or advanced emergency medical technician capable of providing advanced airway management, including endotracheal intubation, intravenous therapy, interpretation and treatment of dysrhythmia, and basic and advanced cardiac and trauma life support. When a physician does not accompany the patient, the transport team must be available to communicate with a healthcare clinician who is capable of providing advice and medical orders if the patient's clinical condition changes. If this is not technically possible, the team should have standing orders to perform acute lifesaving interventions.

C. Minimum Required Equipment During Interfacility Transport

A list of the minimum necessary equipment should be reviewed and discussed regardless of who transports the patient. Ideally, transport packs containing correctly sized equipment and medications for pediatric patients should be prepared in advance and be readily available for emergency situations. **Tables 14-4** through **14-6** list recommended respiratory equipment, transport equipment, and transport medications for pediatric patients. These items should accompany the patient during interfacility transport. A range of equipment and medications may be necessary to cover the pediatric age spectrum. The supplies may be adapted to meet a given patient's clinical needs or mode of transport.

Table 14-4	MINIMUM RECOMMENDED RESPIRATORY EQUIPMENT

- 500-mL resuscitation bag
- 1-liter resuscitation bag with oxygen reservoir
- Oxygen supply tubing: #2
- Manometer with tubing and adapter
- Nasal cannulas: neonatal, pediatric, adult
- Non-rebreather masks: pediatric, adult
- Resuscitation face masks, 1 each: neonate, infant, toddler, pediatric, small adult, large adult
- End-tidal carbon dioxide monitors (disposable):
- Endotracheal tube stylets, 1 each: small, medium, large
- Magill forceps, 1 each: pediatric, adult
- Laryngoscope handles: pediatric, adult
- Extra laryngoscope batteries and light bulbs
- Adhesive tape: 1-inch roll
- Booted hemostat
- 10 mL syringes: 2
- Endotracheal tubes, 2 each: uncuffed 2.0 ,2.5, 3.0, 3.5, 4.0, 4.5 (mm); cuffed 3.0, 3.5, 4.0, 4.5, 5.0, 5.5, 6.0, 6.5, 7.0, 8.0

pediatric, adult
- End-tidal carbon dioxide supplies, 1 each: endotracheal tube adapter, supply tubing, pediatric and adult cannulas
- Positive end-expiratory pressure valve (adjustable)
- Flexible adapters to connect bag-valve system to endotracheal/tracheostomy tube
- Oral airways, 1 each: 5, 6, 7, 8, 9 (pediatric sizing); 0, 1, 2, 3, 4 (adult sizing)
- Nasal trumpets, 1 each: 14F, 16F, 18F, 20F, 22F, 24F, 26F, 30F
- Nebulizer setup with mask and endotracheal tube adapter
- MacIntosh laryngoscope blades: #1, #2, #3, #4
- Miller laryngoscope blades: #0, #1, #2

(mm)
- Water-soluble lubricant
- Suction catheters, 2 each: 6F, 8F, 10F, 12F, 14F
- Tonsil tip suction
- Swivel adapter
- Stethoscope
- Continuous positive airway pressure prong setup with tubing: extra small, small, large
- Scalpel with blade for cricothyrotomy
- Needle cricothyrotomy kit

Table 14-5 MINIMUM RECOMMENDED TRANSPORT EQUIPMENT

- Adhesive tape
- Alcohol swabs
- Arm boards: pediatric, adult
- Arterial line tubing
- Intraosseous needles
- Blood pressure cuffs: neonatal, infant, child, small adult, large adult
- Butterfly needles: 23-gauge, 25-gauge
- Communications backup (eg, cellular telephone)
- Defibrillator electrolyte pads (pediatric and adult) or jelly
- Point-of-care testing capability for glucose
- Electrocardiography electrodes: infant, pediatric, adult
- Flashlights with extra batteries
- Heimlich valve (chest evacuation valve)
- Infusion pumps
- Intravenous fluid tubing: pediatric, adult
- Y tubing for blood administration
- Extension tubing
- Microbore tubing for syringe pumps
- 3-way stopcocks
- Intravenous catheters: 14- to 24-gauge
- Intravenous solutions (plastic bags)
- 1000 mL, 500 mL of normal saline
- 1000 mL of Ringer lactate
- 250 mL of 5% dextrose
- Irrigating syringe (60 mL), catheter tip
- Kelly clamp
- Hypodermic needles, assorted sizes
- Hypodermic syringes, assorted sizes
- Normal saline for irrigation
- Pressure bags for fluid administration
- Pulse oximeter with multiple-site adhesive or reusable sensors: neonatal, pediatric, adult sensors
- Salem sump nasogastric tubes: assorted sizes
- Soft restraints for upper and lower extremities
- Stethoscope
- Suction apparatus
- Suction catheters
- Surgical dressings (eg, sponges, conforming gauze, woven gauze)
- Tourniquets for venipuncture/intravenous access
- Scissors
- Spinal and cervical spine immobilization devices
- Thermometer
- Sterile and nonsterile gloves
- Chest tubes: 12F, 16F, 20F, 26F
- Chest tube insertion kits
- Nasogastric tubes: 8F, 10F, 12F, 14F
- Urinary Foley catheters and urinary collection bags
- Clear dressings for intravenous catheter sites

Table 14-6 MINIMUM RECOMMENDED TRANSPORT MEDICATIONS

- Pediatric emergency medication reference guide
- Adenosine
- Albuterol
- Amiodarone
- Atropine
- Calcium chloride
- Topical anesthetic spray: dermal, oral mucous membrane
- Lidocaine, 2 g/10 mL
- Magnesium sulfate
- Mannitol
- Methylprednisolone
- Metoprolol
- Naloxone
- Narcotic analgesics (eg, morphine, fentanyl)

- Dextrose 25%
- Dextrose 50%
- Digoxin
- Diltiazem
- Diphenhydramine
- Dopamine
- Epinephrine/adrenaline, 1 mg/10 mL (1:10,000)
- Epinephrine/adrenaline, 1 mg/1 mL (1:1000) multiple-dose vial
- Fosphenytoin (must be refrigerated)
- Furosemide
- Glucagon
- Heparin, 1000 units/1 mL
- Isoproterenol
- Labetalol
- Lidocaine, 100 mg/10 mL
- Neuromuscular blocking agents (eg, vecuronium, pancuronium, rocuronium)
- Nitroglycerin injection
- Nitroglycerin tablets
- Nitroprusside
- Normal saline, for injection
- Phenobarbital
- Potassium chloride
- Procainamide
- Prostaglandin E_1 (must be refrigerated)
- Pulmonary surfactant
- Sedatives/hypnotics (eg, lorazepam, midazolam, ketamine)
- Sodium bicarbonate
- Sterile water, for injection
- Terbutaline

D. Monitoring During Interfacility Transport

At a minimum, interfacility transport must provide the same level of monitoring as in the intrafacility process to mimic the intensive care unit setting (**Table 14-1**). Some patients may benefit from arterial blood pressure, central venous pressure, intracranial pressure, and/or end-tidal carbon dioxide monitoring. For those who require mechanical ventilatory support, the position of their endotracheal tubes should be noted and secured prior to transport. The adequacy of oxygenation and ventilation must be reconfirmed frequently. The mode of mechanical ventilation must be evaluated before transport to ensure acceptability and stability during travel. The child's status, interventions, and management during transport must be documented in the patient medical record. The receiving facility should be given copies of all transport documentation along with the information from the referring institution.

Key Points

Transport of the Critically Ill Child

- Staff at the referring hospital should carry out all interventions to stabilize a pediatric patient, including performing or assisting the transport team in any procedures necessary to prevent clinical deterioration during transport.
- To allow safe transport, patients should be transported only when sufficiently stabilized.
- Adequate information is required to determine a patient's optimal mode of transport and the composition of the transport team.
- Early contact must be established with the receiving physician and facility to ensure the most expedient transfer of a patient.

- Whenever possible, all appropriate materials (ie, medical record, laboratory results, radiographic studies, and contact phone numbers) should be prepared prior to transport.
- The transport team and receiving physician/facility should be informed about any changes in the clinical condition of the patient.
- The appropriate equipment must accompany a patient during all inter- and intrafacility transports.
- The referring transport team should provide all necessary equipment and medications, including sufficient materials to sustain the patient for a minimum of 30 minutes beyond the expected transport time.

Suggested Readings

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NEUROLOGIC EMERGENCIES

Objectives

- Recognize the normal physiology of the intracranial vault and cerebral blood flow.
- Review the emergent neurologic assessment of the pediatric patient.
- Summarize the acute management of seizures and status epilepticus.
- Describe the approach to the patient with altered mental status or coma.
- Outline the current management of intracranial hypertension.

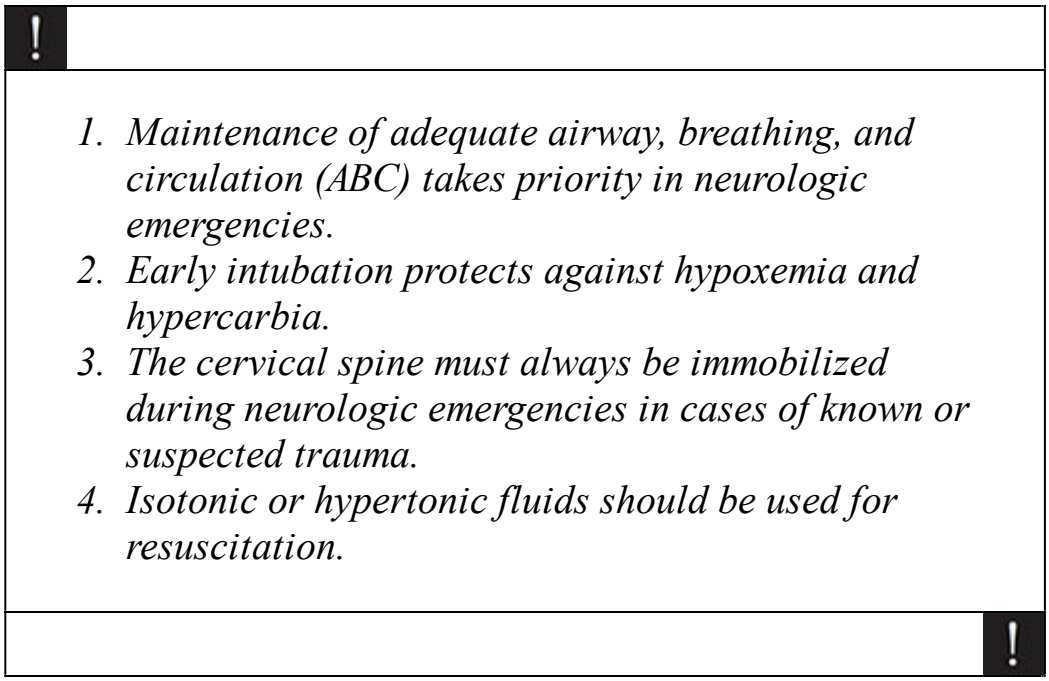
I. INTRODUCTION

A pediatric patient who presents with an altered level of consciousness represents both an emergency and diagnostic challenge. The differential diagnosis may be extensive, and basic resuscitative efforts must be performed while pursuing a diagnosis. A basic understanding of the physiology of the central nervous system (CNS) is essential in recognizing and managing these patients. Many compensatory mechanisms serve to preserve cerebral function and neuronal survival under pathophysiologic conditions; examples include displacement of cerebrospinal fluid (CSF) to the spinal subarachnoid space and along the cerebrospinal axis and compression of the venous bed. Functional recovery requires resolving the insult promptly and minimizing secondary injury. Thus, maintenance of normal oxygen delivery to the CNS remains a primary goal in patients with neurologic injury.

Prevention of secondary injury continues to be the paramount goal of neurointensive care.

Establishing a patent airway and effective breathing and circulation (ABC) takes priority in neurologic emergencies, as in any other emergent situation. However, in neurologic emergencies, any known or suspected trauma requires cervical spine stabilization during airway manipulation (**Chapters 2 and 9**).

Patients with neurologic disease may suffer respiratory insufficiency for a variety of reasons, including hypoventilation and airway obstruction. Deteriorating neurologic status raises the possibility of impending respiratory failure, and the establishment of an artificial airway should be promptly considered. Generally, elective intubation prevents hypoxemia and hypercarbia and is safer than emergent intubation. Finally, isotonic or hypertonic fluids should be used for resuscitation to minimize the risk of cerebral edema.



1. *Maintenance of adequate airway, breathing, and circulation (ABC) takes priority in neurologic emergencies.*
2. *Early intubation protects against hypoxemia and hypercarbia.*
3. *The cervical spine must always be immobilized during neurologic emergencies in cases of known or suspected trauma.*
4. *Isotonic or hypertonic fluids should be used for resuscitation.*

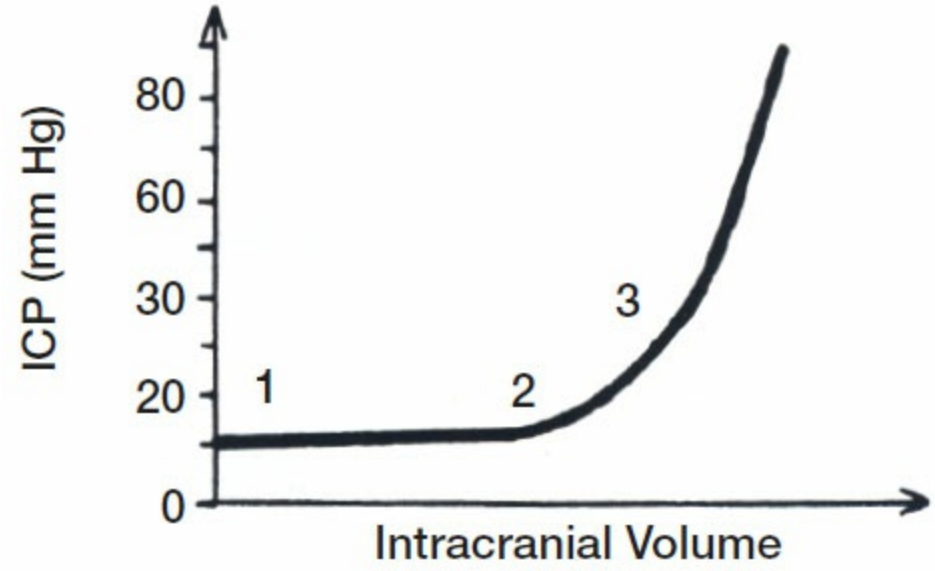
II. BASIC PHYSIOLOGY OF THE INTRACRANIAL VAULT

Many neurologic emergencies share common pathophysiologic processes that affect the pressure within the cranial vault and/or impair cerebral blood flow (CBF). The human body has a number of regulatory mechanisms that help to reduce these changes and their impact on neurologic function. A basic understanding of these mechanisms, their derangements, and the methods of improving them is imperative for the treatment of children with neurologic conditions.

A. Intracranial Pressure

The Monro-Kellie doctrine states that the cranial vault contains a fixed volume consisting of 3 basic components—brain (80%), blood (10%), and CSF (10%)—encased by the thick, inelastic dura mater and the semirigid cranium. These components exist in a state of volume-pressure equilibrium, such that expansion of one induces a reduction in the volume of one or both of the others. **Figure 15-1** illustrates intracranial compliance. The interaction of these 3 components and the physiologic mechanisms controlling each of them provides the framework for neurointensive care.

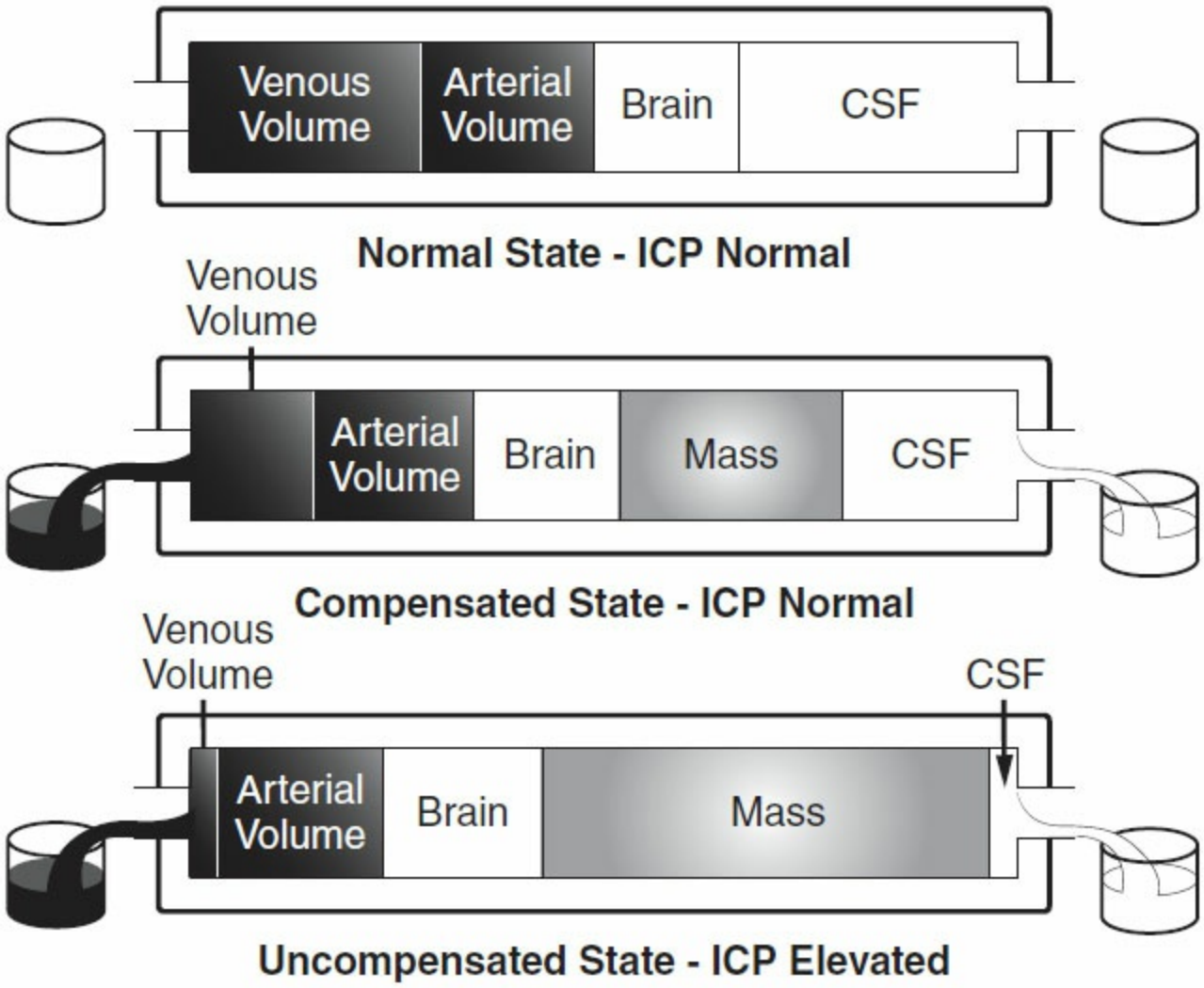
Figure 15-1. Intracranial Compliance



Between points 1 and 2, although intracranial volume is increased (ie, by tumor, edema, or hemorrhage), the intracranial contents remain at a constant pressure. Displacement of cerebrospinal fluid and intracerebral blood into the spinal space accommodates this increased volume. At point 2, although the intracranial pressure (ICP) is normal, any further increases in volume (eg, tumor, edema, obstructive hydrocephalus, or intracranial hemorrhage) will produce an exponential rise in ICP, which may be life-threatening. Point 3 represents a decompensated state and a neurosurgical emergency with dangerously high ICP.

Infants with open fontanelles may be prone to intracranial hypertension because the inelastic dura mater, which encases the brain, limits expansion of the intracranial compartment. Furthermore, infants have a shorter craniospinal axis (measured from the cranial dura down the length of the spinal canal to the lumbosacral area) than the adult; this provides less space to allow for the displacement of CSF or cerebral blood volume (CBV). **Figure 15-2** demonstrates how the intracranial compartment can compensate for a change in the volume of a component.

Figure 15-2. Interactions Among Intracranial Compartments



The top panel illustrates the 3 components of the intracranial vault in equilibrium with normal intracranial pressure (ICP). The middle panel represents an increasing mass lesion (eg, hemorrhage); the compensatory mechanisms include the extrusion of cerebrospinal fluid (CSF) and venous blood volume. This corresponds to a move from point 1 to point 2 in **Figure 15-1**, and the ICP remains normal. The bottom panel illustrates the uncompensated state, which occurs following maximal compensatory fluid displacement from the intracranial space, producing an elevated ICP. This corresponds to a move from point 2 to point 3 in **Figure 15-1**. The interaction of these 3 components and the physiologic mechanisms controlling each provide the framework for neurointensive care.

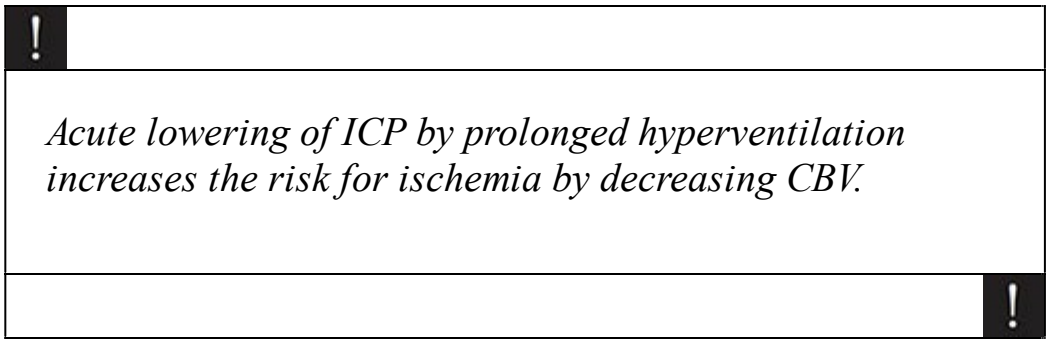
1. The Brain

The brain constitutes the largest volume (80%) of the intracranial vault. The brain parenchyma is composed of neurons (50%) and glial and vascular elements. Neurons are electrically active cells requiring a significant amount of energy to produce neurotransmitters. However, because the brain contains only minimal energy stores, increases in CBF must occur in response to increased metabolic needs, such as during seizures and hyperthermia.

The brain's volume is dynamic and may respond to cellular edema or an increase in the extracellular fluid space. Cellular injury produces edema (cytotoxic edema) due to a failure of the mechanisms that maintain cellular homeostasis. Cytotoxic edema responds particularly poorly to osmotic therapy and often indicates a poor prognosis. The extracellular fluid volume increases with any process that increases the blood-brain barrier permeability (vasogenic edema). Both forms of edema contribute to increased brain volume, and thus increased intracranial pressure (ICP), in many pathological processes.

2. Cerebral Blood Volume

Blood in arteries, veins, and capillaries occupies approximately 10% of the intracranial space. CBF changes in response to alterations in brain metabolism, blood pressure, and arterial $Paco_2$ and Pao_2. Arteriolar tone regulates these changes, so CBF must be altered to effect changes in CBV. Although increased arteriolar tone (eg, by decreasing $Paco_2$ via hyperventilation) produces a rapid decrease in CBV (and consequently ICP), this places the brain at risk for ischemic injury. Therefore, recommendations support only brief manipulation of $Paco_2$ to reduce CBV and ICP.



3. Cerebrospinal Fluid

CSF accounts for approximately 10% of the intracranial volume. The brain essentially floats in the CSF, which provides a protective cushion and prevents traction on nerve roots, blood vessels, and delicate membranes. The intraventricular volumes are approximately 40 to 60 mL in infants, 60 to 100 mL in young children, 80 to 120 mL in older children, and 100 to 160 mL in adults. The choroid plexus produces approximately 70% of the CSF. The remainder is formed in the extrachoroidal sites, including the ventricular ependyma, sylvian aqueduct, subarachnoid pial surface, and brain and spinal cord parenchyma. The rate of formation is approximately 0.35 to 0.40 mL/min, or 500 to 600 mL/day, independent of age. The turnover time for CSF is 5 to 7 hours.

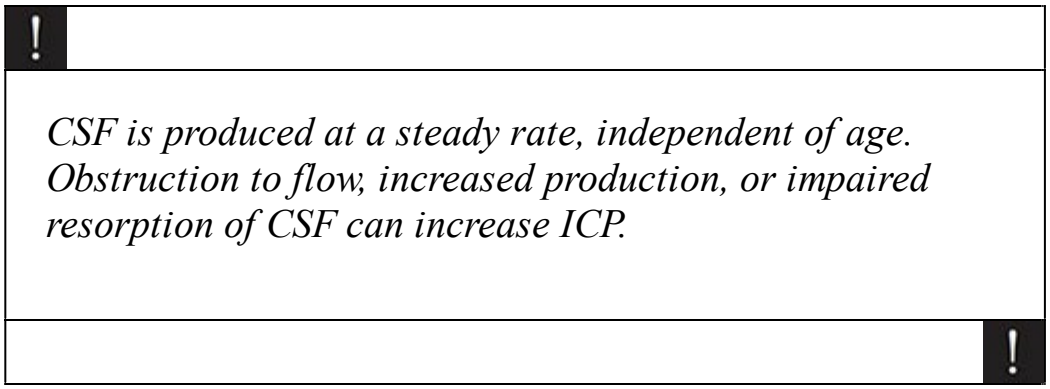
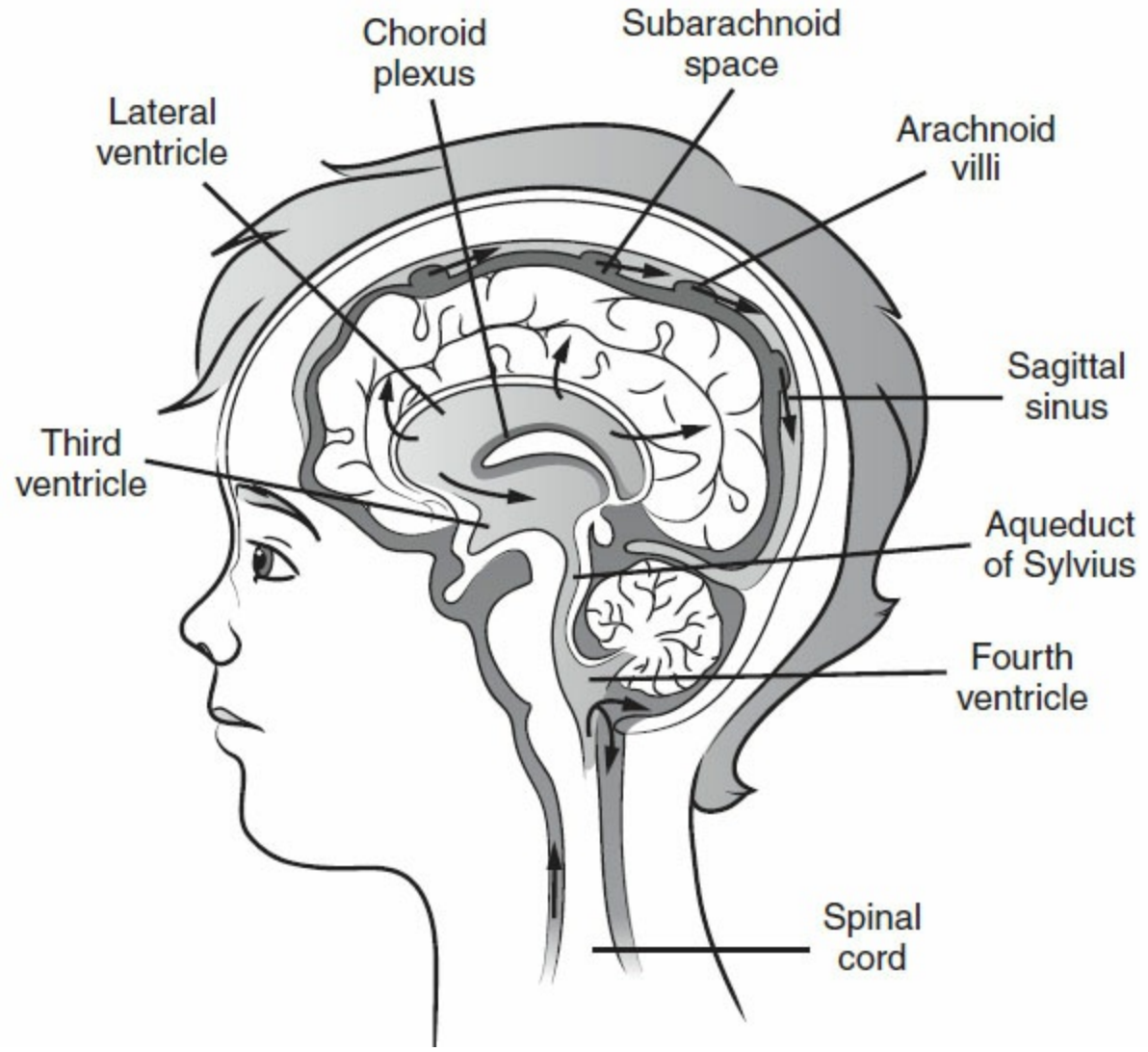

!
CSF is produced at a steady rate, independent of age. Obstruction to flow, increased production, or impaired resorption of CSF can increase ICP.
!

Figure 15-3 shows the normal pathway of CSF flow, which is generated by pressure gradients and cilia on the ependymal surfaces. The arachnoid villi on the superior surface of the brain resorb the CSF and drain into the superior sagittal sinus.

Figure 15-3. Flow of Cerebrospinal Fluid



Increased production, impaired resorption, or obstructed flow can all increase CSF volume and ultimately ICP. CSF provides a chemically appropriate environment for neurotransmission and the removal of metabolic by-products. As an ultrafiltrate of plasma, CSF contains the same electrolyte concentrations as serum and the rest of the extracellular fluid. Glucose and other larger molecules are transported into the CSF across the impermeable blood-brain barrier, so alterations in the barrier can disrupt neurologic function. The use of osmotically active substances to reduce brain water in the treatment of intracranial hypertension requires an intact blood-brain barrier.

B. Cerebral Blood Flow

The brain depends entirely upon exogenous glucose for its metabolic needs, and CBF is tightly matched to metabolic demands. Not surprisingly, the ratio of CBF to gray matter (active neurons) exceeds that to white matter, which is less metabolically active. Other factors affecting CBF include mean arterial pressure, $Paco_2$, Pao_2, and age. The normal adult CBF is 50 mL/100 g/min, whereas it is 40 mL/100 g/min in neonates and 100 mL/100 g/min in children. The critical threshold below which ischemia develops, producing electroencephalographic (EEG) changes, is 20 mL/100 g/min in the adult and 5 to 10 mL/100 g/min in the infant.

As with all organs, the perfusion pressure is determined by the difference between the upstream and downstream pressures. In the brain, either the central venous pressure (CVP) or the ICP may provide

the greater downstream pressure. Therefore, cerebral perfusion pressure (CPP) = mean arterial pressure (MAP) – (ICP or CVP), whichever is higher.

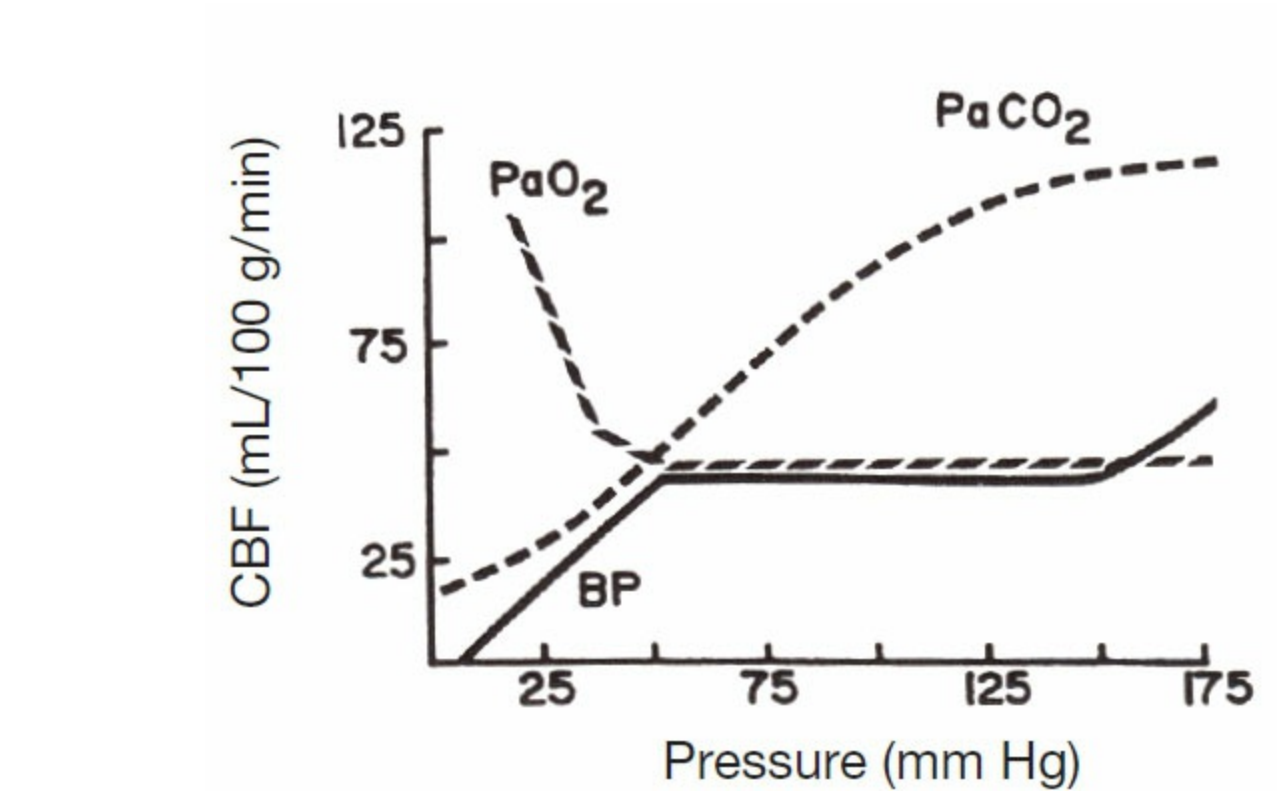
!

1. *Chronic hypertension shifts the autoregulatory curve rightward, so an acute drop in MAP >25% from the baseline may place the patient at risk of ischemia.*
2. *Hypercarbia and hypoxia both dilate cerebral blood vessels, leading to increased CBF and possibly ICP.*
3. *Increased brain activity (including seizures) increases CBF and possibly ICP.*

!

The body maintains a stable CBF over a wide range of arterial pressures through autoregulation, producing dilation or constriction of the precapillary arterioles, as necessary. **Figure 15-4** depicts autoregulation in a normal adult. At extremely low CPP, CBF decreases and ischemia ensues. At CPP above the autoregulatory range, high CBF produces cerebral edema. With time, the range over which autoregulation occurs adjusts to the patient's normal blood pressure. Generally, the lower limit for autoregulation is 25% below the patient's baseline MAP, and this should be the maximal induced reduction in blood pressure used to decrease the risk of cerebral ischemia. Due to the lower prevailing MAP in young children and infants, the acceptable CPP is slightly lower than the value targeted in adults (60-70 mm Hg), but the optimal value has not been scientifically determined.

Figure 15-4. Autoregulation of Cerebral Blood Flow



CBF, cerebral blood flow

CBF correlates linearly with $Paco_2$, ranging from 20 to 80 mm Hg (2.7-10.7 kPa). The changes in CBF with altered $Paco_2$ occur independently of a change in arterial pH. Hyperventilation to lower $Paco_2$ provides an effective means of rapid reduction in CBV and, consequently, ICP. However, as noted previously, this may place the patient at risk of cerebral ischemia. Hypoxemia below 40 to 50 mm Hg (5.3-6.7 kPa) also dilates the cerebral blood vessels, resulting in increased CBF to maintain cerebral oxygen delivery to areas of very low levels of oxygen content. However, this occurs at the expense of increased CBV and possibly increased ICP. Finally, anything that increases the metabolic demands of the brain, including hyperthermia and seizures, increases CBF.

III. EMERGENT NEUROLOGIC ASSESSMENT OF THE PEDIATRIC PATIENT

A. Level of Consciousness

The neurologic examination in emergencies is focused on assessing the level of consciousness and signs of intracranial hypertension or focal pathology. Certain aspects of the exam cannot be assessed in young children due to their inability to follow commands or communicate verbally. Careful serial assessment of the level of consciousness is the most important component of this exam. Combative behavior may be a sign of impaired neurologic function. A quiet child with eyes closed may be asleep or may have depressed consciousness. The response to noxious stimuli in such patients often provides clues to the degree of altered consciousness. The Glasgow Coma Scale (GCS) score is widely used in adult patients, and a modified pediatric scale exists for infants and children (**Table 15-1**).

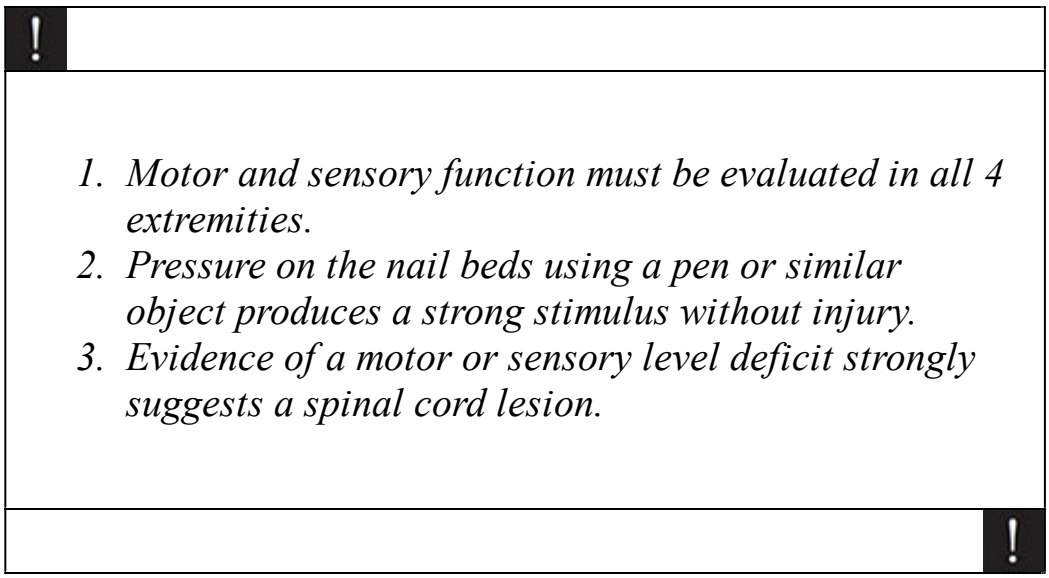
Table 15-1 GLASGOW COMA SCALE MODIFIED FOR INFANTS AND CHILDREN

Clinical Parameter	Infants (Ages 0-12 Months)	Children (Ages 1-5 Years)	Points[a]
Eye opening	Spontaneous	Spontaneous	4
	Response to speech	Response to speech	3
	Response to pain	Response to pain	2
	No response	No response	1
Verbal response	Coos/babbles	Appropriate words	5
	Irritable cries	Inappropriate words	4
	Cries	Persistent cry	3
	Moans	Grunts	2
	No response	No response	1
Best motor response	Normal	Spontaneous	6
	Withdraws to touch	Localized pain	5
	Withdraws from pain	Withdraws from pain	4
	Flexor response	Flexor response	3
	Extensor response	Extensor response	2
	No response	No response	1

[a]Total Glasgow Coma Scale score = eye + verbal + motor points; best possible score = 15; worst possible score = 3.

B. Movement

Careful observation for asymmetric movements of the extremities and facial/ocular muscles may also provide diagnostic clues. Abnormal movements may indicate seizures (clonic, stereotyped), toxins or metabolic disease (tremors, asterixis, myoclonus), or basal ganglia injury (chorea, dystonia). Abnormal postures in response to stimulation, including that from suctioning and other procedures, consist of flexor posturing, extensor posturing, and flaccidity. All 4 extremities should be checked for response by applying pressure with a pen or similar object across the nail bed. Appropriate responses indicate intact sensory and effector pathways. The terms *decorticate posture* and *decerebrate posture* are oversimplifications, but progression along this spectrum generally indicates more severe or more caudal lesions.



1. *Motor and sensory function must be evaluated in all 4 extremities.*
2. *Pressure on the nail beds using a pen or similar object produces a strong stimulus without injury.*
3. *Evidence of a motor or sensory level deficit strongly suggests a spinal cord lesion.*

In cooperative patients, careful assessment for a motor or sensory level response may pinpoint the location of any spinal cord lesion.

C. Motor and Sensory Evaluation

Brainstem reflexes (cranial nerve exam) allow for localization of the lesion, provide diagnostic clues, and indicate whether the airway reflexes and respiratory drive remain intact. Pupillary, corneal, oculocephalic (doll's eye), vestibular (cold-water calorics), and gag reflexes should be assessed in patients with depressed consciousness to help determine the location and severity of the lesion. In patients with possible cervical spine injury, oculocephalic reflexes should not be tested.

In patients requiring neuromuscular blockade, pupillary reactions may constitute the entire neurologic exam. Pupillary reactivity can persist in deep coma caused by metabolic disease, but its absence indicates a lesion of the third cranial nerve or midbrain, including possible herniation syndromes. An absent or diminished gag reflex places the patient at increased risk of airway obstruction and aspiration.

D. Respiratory Patterns

Respiratory patterns may indicate specific lesions or the need to secure an artificial airway. In Cheyne-Stokes respiration, a gradually increasing period of hyperventilation diminishes to

hypoventilation and apnea in a regularly alternating pattern. Sustained hyperventilation (with large tidal volumes) indicates rostral brainstem injury. This must be distinguished from the hyperventilation induced by pulmonary edema, which may also be caused by neurologic disease (neurogenic pulmonary edema). Patients with true central hyperventilation have elevated (or at least normal) arterial Pao_2 and increased arterial pH. On the other hand, patients may have Kussmaul respirations (large, rapid breaths) as compensation for a metabolic acidosis (such as with diabetic ketoacidosis). Caudal lesions produce apneustic breathing, which consists of inspiratory pauses, typically lasting 2 to 3 seconds. Ataxic breathing, the irregular alteration between deep and shallow breathing, occurs with lesions of the medulla. The lower the lesion, the more likely the patient will require mechanical ventilation, and patients with apneustic or ataxic breathing have a high likelihood of requiring complete mechanical respiratory support.

E. Herniation Syndromes

With herniation of CNS components into another compartment, several discrete syndromes may be seen, any of which indicate a poor prognosis and require immediate intervention for recovery (**Figure 15-5**).

Figure 15-5. Herniation Syndromes

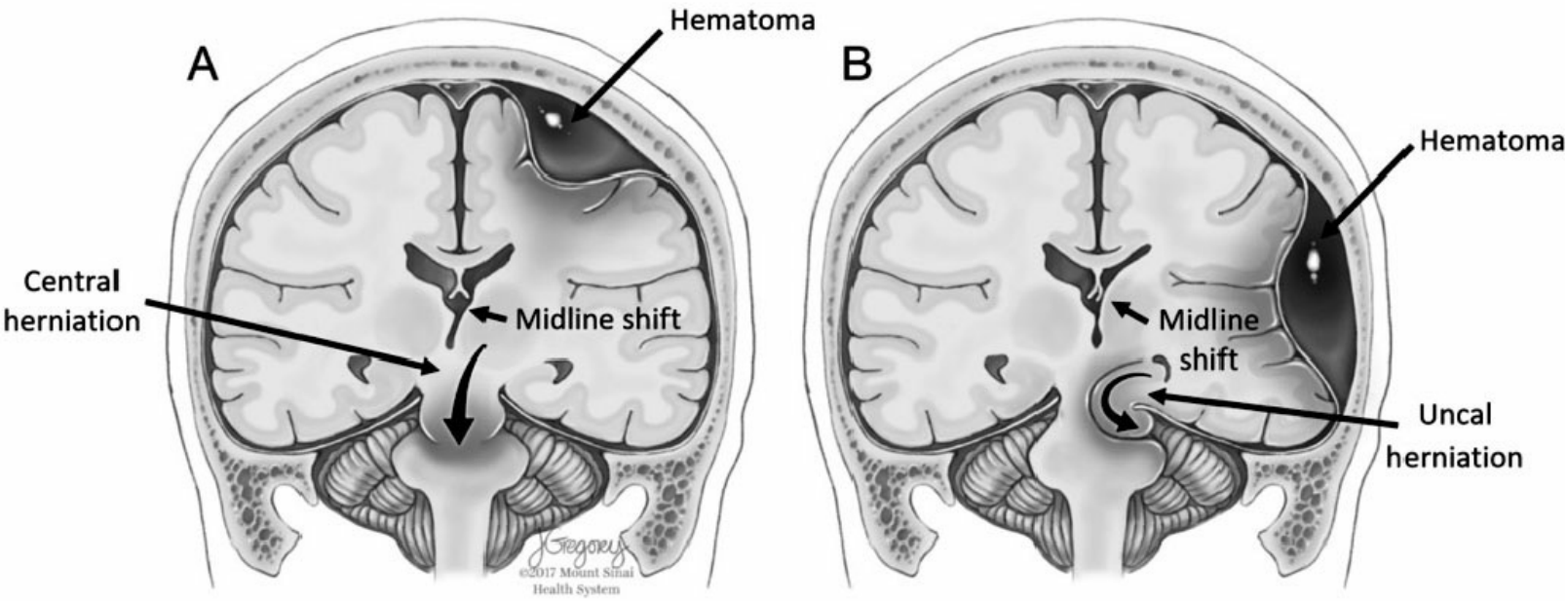
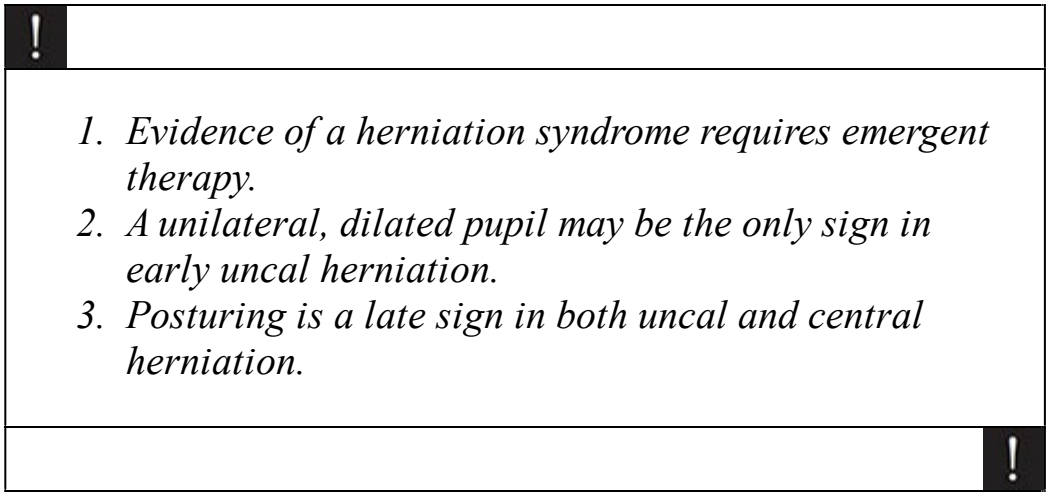


Illustration by Jill K. Gregory, CMI. Reprinted with permission from Mount Sinai Health System, New York, NY.

A. Central herniation occurs when the hemispheres and basal nuclei are displaced downward through the tentorial notch. Initially, patients have depressed or altered consciousness, Cheyne-Stokes respirations, and small, reactive pupils. The eyes usually remain conjugate at this stage. With progression, patients may develop flexor posturing of the upper extremities. Further progression produces central hyperventilation, fixed mid-position pupils, and extensor posturing often confused with seizures. At this stage, patients may also have disconjugate eye movements in response to oculocephalic testing or cold-water calorics.

B. Uncal herniation occurs when a lateral lesion pushes the medial uncus and hippocampal gyrus over

the lateral edge of the tentorium. The most consistent early sign is the unilaterally dilated pupil. Patients with uncal herniation may progress in an extremely rapid fashion to depressed consciousness with extensor then flexor posturing, typically of the contralateral extremities. They also develop disconjugate gaze.



1. *Evidence of a herniation syndrome requires emergent therapy.*
2. *A unilateral, dilated pupil may be the only sign in early uncal herniation.*
3. *Posturing is a late sign in both uncal and central herniation.*

IV. STATUS EPILEPTICUS

Case Study

A 5-year-old boy who had been previously well presents with a 2-day history of fever to 40°C (104°F) and complaining of seeing bugs on the walls of his room. He is irritable and combative on arrival. He begins to have a generalized tonic-clonic seizure. You are called to help manage this patient.

Detection

– What is the most likely diagnosis?

Intervention

– Which, if any, antiepileptic medication(s) should be administered?

Reassessment

– When, if at all, would you consider a second antiepileptic medication?

– What are the indications for an imaging study?

– What are the indications for performing a lumbar puncture?

Effective Communication

– Where is the best place to manage the care of this patient?

– Which subspecialty would you consult?

Teamwork

– How are you going to implement the treatment strategy?

Status epilepticus (SE) is a common pediatric neurologic emergency. It is estimated to affect between 25,000 and 50,000 children annually, with 40% occurring in children younger than 2 years of age. Etiologies of SE are summarized in **Table 15-2**.

Table 15-2	ETIOLOGY OF STATUS EPILEPTICUS IN PEDIATRIC PATIENTS
Antiepileptic noncompliance	Drug toxicity (penicillin, ciprofloxacin, theophylline, cyclosporine, amitriptyline, phenothiazines, lidocaine, imipenem, tacrolimus, cocaine, sympathomimetics, isoniazid, ethanol)
Cerebrovascular accidents (stroke, AVM)	Fever
CNS infections (meningitis, encephalitis)	Drug withdrawal
CNS tumors	Malignant hypertension
Head trauma (accidental, nonaccidental)	Underlying CNS disorders
Hypoxic-ischemic injury	Neurocutaneous syndromes (Sturge-Weber, tuberous sclerosis, NF1)
Electrolyte disorders (glucose, sodium, calcium	

AVM, arteriovenous malformations; CNS, central nervous system; NF1, neurofibromatosis type 1

The current operational definition of generalized convulsive SE in adults and older children (>5 years) refers to ≥5 minutes of continuous seizure or ≥2 discrete seizures between which there is incomplete recovery of consciousness. The authors of this definition cite a paucity of pediatric data to allow recommendations in children younger than 5 years. Although there is general agreement that seizures continuing for longer than 5 minutes should usually be treated, overaggressive treatment may lead to avoidable morbidity.

A. Pathophysiology and Organ Dysfunction

Seizures result from abnormal synchronous electrical discharges (depolarization) of a group of neurons in the CNS. Depolarization stems from the influx of sodium into the neuron, whereas repolarization results from the efflux of potassium from the cell, which restores the resting negative electrical potential. This potential is regulated by a sodium-potassium pump that is driven by adenosine triphosphate.

Prolonged seizures may cause selective neuronal loss in the hippocampus, cortex, and thalamus, areas rich in glutamate receptors. This calcium-mediated neuronal cell death is referred to as the

excitotoxic theory, which is similar to that proposed to occur in CNS ischemia. Although prolonged seizures may be sufficient to cause neuronal damage, the superimposition of hypoxia, hypotension, acidosis, and hyperpyrexia exacerbate the degree of damage.

SE may be divided into 2 stages. The first 30 minutes are characterized by increased autonomic activity: hypertension, tachycardia, hyperglycemia, diaphoresis, and hyperpyrexia. This is followed by a second stage, characterized by multiorgan involvement, and includes respiratory failure (hypoxemia, hypercarbia), decreased CBF, increased ICP, and a dropping arterial blood pressure. Severe acidosis ensues, and the patients may develop leukocytosis, hyperkalemia, and an elevated creatine kinase (secondary to increased muscle activity). The characteristics of early and late SE are compared in **Table 15-3**.

Table 15-3 PHYSIOLOGIC DERANGEMENTS IN EARLY AND LATE STATUS EPILEPTICUS

	Early (<30 Minutes)	Late (>30 Minutes)	Complication
Blood pressure	↑	↓	Hypotension
Pao_2	↓	↓	Hypoxia
$Paco_2$	↑	Variable	↑ ICP
Serum pH	↓	↓	Acidosis
Autonomic	↑	↔	Arrhythmias
CK	Normal	↑	Renal failure
K^+	Normal	↑	Arrhythmias
CBF	↑ 900%	↑ 200%	CNS bleeding
$CMRo_2$	↑ 300%	↑ 300%	Ischemia

CK, creatine kinase; CBF, cerebral blood flow; $CMRo_2$, cerebral metabolic rate of oxygen consumption; ICP, intracranial pressure; CNS, central nervous system

Adapted with permission. © 1987 Wolters Kluwer Health. Dean JM, Singer HS. Status epilepticus. In: Rogers MC, ed. *Textbook of Pediatric Intensive Care Medicine*. Baltimore, MD: Williams & Wilkins; 1987:618.

A relative decrease in CBF occurs at a time when demands for cerebral energy substrates (oxygen, glucose) are markedly increased and cerebral autoregulation is depressed. This signals the failure of adaptive mechanisms.

Respiratory failure associated with SE may be due to increased CO_2 production from the hypermetabolic state, decreased respiratory drive (due to muscle fatigue or medications used to suppress the seizure), and increased mechanical load on the respiratory muscles. Concomitant aspiration or neurogenic pulmonary edema is also possible and can contribute to both hypoxemia and respiratory acidosis.

The cardiovascular changes and initial hyperadrenergic state are due to the release of endogenous catecholamines. Patients have an initial increase in systemic vascular resistance that will decrease over time. As the seizure continues, hyperpyrexia must be prevented because it contributes to neuronal cell death. Studies in adult patients have shown that a CSF pleocytosis is possible; however, any elevation in the CSF cell count in a pediatric patient is suggestive of possible infection.

Appropriate cultures should be obtained and therapy instituted. The prolonged muscle activity may lead to elevation in creatine kinase and production of myoglobin with subsequent myoglobinuria; therefore, careful attention to the patient's hydration status and renal function is essential. The massive catabolic stress of SE may also lead to hyperkalemia, so attention to electrolyte status is required. Acidemia may be worsened by impaired ventilation, hypoxemia, and anaerobic metabolism.

B. Evaluation and Management of Status Epilepticus

The therapeutic goals for SE include: (1) general supportive care, (2) termination of SE, (3) prevention of seizure recurrence, (4) correction of the precipitating causes, and (5) prevention and treatment of potential complications. Immediate attention to the airway, breathing, and circulation is essential. Status epilepticus requires that management and evaluation be performed simultaneously. A suggested clinical approach to SE is outlined in **Table 15-4**.

Table 15-4 TREATMENT ALGORITHM FOR STATUS EPILEPTICUS

1. Assess airway, breathing, and circulation. Apply oxygen, establish airway and ventilation. Reassess throughout management. Consider airway adjuncts: nasopharyngeal tube and F_{IO_2} 1.0 via non-rebreather. Anticipate need for endotracheal intubation.
2. Establish IV or IO access. Obtain initial laboratory studies. Administer dextrose if patient hypoglycemic, isotonic fluid boluses for inadequate perfusion and/or hypotension
3. Pharmacotherapy
   a. Administer lorazepam, 0.1 mg/kg IV (maximum 4 mg); may repeat 1 time.
   Alternatives: diazepam, 0.5 mg/kg PR; midazolam, 0.2 mg/kg IM.

   b. Administer fosphenytoin, 20 mg/kg IV, no faster than 150 mg PE/min immediately after initial dose of benzodiazepine. Consider an additional 5-10 mg/kg of fosphenytoin prior to start of barbiturates if seizures continue. Assess and monitor airway and hemodynamics.
   Alternative: phenytoin, 20 mg/kg, no faster than 1 mg/kg/min with a maximum rate of 50 mg/min.
   Anticipate need for endotracheal intubation.
   Employ continuous cardiopulmonary monitoring.

   c. If seizures persist after initial benzodiazepine and fosphenytoin, consult neurology and administer **PHENobarbital**, 20 mg/kg IV,
   no faster than 1 mg/kg/min up to a maximum rate of infusion 50 mg/min. Monitor blood pressure and consider elective endotracheal intubation.
   Anticipate need for endotracheal intubation.
   Employ continuous cardiopulmonary monitoring.

   d. If seizures persist, initiate refractory SE treatment: midazolam bolus of 0.2 mg/kg and midazolam infusion beginning with
   0.1 mg/kg/h, increased every 15 min until seizure activity stops. Monitor respiratory status, ECG, blood pressure, and EEG.
   Anticipate need for endotracheal intubation.
   Employ continuous cardiopulmonary monitoring.

   e. If seizures persist, administer **PENTobarbital**, 5 mg/kg IV, up to a maximum rate of 50 mg/min. Begin infusion at 1 mg/kg/h. Additional boluses of 5 mg/kg may be given to achieve either burst suppression pattern on EEG or cessation of seizure activity. Monitor vital signs closely.
   Anticipate need for endotracheal intubation.
   Employ continuous cardiopulmonary monitoring.
   Other pharmacologic agents used for refractory SE include valproic acid (20 mg/kg IV) or levetiracetam (20-30 mg/kg IV load)

f. Clinical seizure activity may cease, but nonconvulsive SE may persist. EEG evaluation is warranted in patients who do not return to baseline neurologic status.

4. Diagnostic evaluation. Treat underlying causes and systemic complications.

PR, per rectum; ED, emergency department; IV, intravenous; IO, intraosseous; IM, intramuscular; PE, phenytoin sodium equivalents; SE, status epilepticus; ECG, electrocardiogram; EEG, electroencephalogram

Although most seizures are self-limited and stop in ≤5 minutes, most children who arrive to the emergency department or pediatric ICU have been seizing for a significant length of time. This may include time seizing before discovery, time waiting for emergency personnel, and time spent en route to the hospital. Patients may have already received medications from family members or emergency medical personnel, which may contribute to any respiratory depression seen on arrival.

!

Management of SE includes:

1. *General supportive care*
2. *Termination of SE*
3. *Prevention of seizure recurrence*
4. *Correction of precipitating causes*
5. *Prevention and treatment of potential complications*

!

1. Airway Management

The immediate assessment of the airway begins with a check for patency and reflexes. The airway may be secured by simple maneuvers, such as suctioning, positioning, and the use of airway adjuncts (nasal trumpet). High-flow oxygen should be administered and pulse oximetry instituted to evaluate oxygenation status. The presence of nasal flaring, poor chest rise, retractions, cyanosis, abdominal paradoxical breathing, diminished breath sounds, or apnea suggests that the patient should be endotracheally intubated and mechanically ventilated. If the patient requires the administration of a neuromuscular blocking agent for intubation, one with a short half-life should be used. This will stop the motor component of the seizure but will not abort the underlying electrical activity. Patients who are under continued paralysis should be monitored with continuous EEG.

2. Cardiovascular Management

Tachycardia, cool extremities, delayed capillary refill, diminished pulses, and poor urine output suggest hypovolemia. The resulting poor perfusion may be exacerbated by the addition of medications and positive pressure ventilation. The pediatric advanced life support guidelines should be followed to obtain vascular access, through which the patient may receive isotonic fluid boluses (20 mL/kg). Antipyretics should be administered early in the management of the patient in SE.

3. Investigations

Specific studies should be tailored to match the patient history; however, serum glucose should be checked immediately, particularly in young infants. Other blood work may include testing serum electrolytes (sodium, potassium, calcium, and magnesium) and liver function, measuring arterial blood gas and antiepileptic drug levels, and ordering urine toxicology screening. In patients with altered mental status and signs of focality, an imaging study (noncontrast computed tomography [CT] scan) should be considered prior to a lumbar puncture for CSF examination. Antibiotics and antivirals (if indicated) should be promptly administered and the lumbar puncture delayed in the event that there are severe respiratory, cardiovascular, or other contraindications, such as a coagulopathy or elevated ICP.

Nonconvulsive SE is possible in patients who do not quickly return to baseline. Electrographic seizure activity has been reported in up to 15% of patients whose overt clinical seizures are pharmacologically controlled. This form of SE is defined by EEG criteria and presents with altered mentation and absent or subtle motor findings (ie, finger twitch). The incidence of nonconvulsive SE in pediatrics is unknown, but adult studies have shown a mortality rate between 30% and 50%.

C. Pharmacotherapy

The goal of administering an antiepileptic drug is to achieve rapid and safe termination of the event and to prevent recurrence. Common first-line medications are benzodiazepines (diazepam, lorazepam, and midazolam), and second-line agents include phenytoin/fosphenytoin and phenobarbital. The pharmacology and routes of administration of these agents are presented in **Table 15-5**. Common errors in the management of SE include insufficient drug dosages, delay in advancing to a second-line drug, and inadequate supportive care.

Table 15-5 PHARMACOLOGIC AGENTS TO TREAT STATUS EPILEPTICUS

Drug	Dosage	Onset	Duration
Lorazepam	0.1 mg/kg/dose IV/IO; may repeat once Maximum: 4 mg/dose	2-3 min	>6 h
Midazolam	0.05-0.2 mg/kg/dose IV/IO/IM Maximum: 5 mg/dose Infusion: 1 µg/kg/min (range, 1-18 µg/kg/min) PR: 0.5-1 mg/kg/dose	2-5 min	30-60 min
Diazepam	0.1-0.3 mg/kg/dose Maximum: 10 mg/dose PR: 0.3-0.5 mg/kg/dose	2-5 min	60-90 min
Fosphenytoin	20 mg/kg load IV/IM PE 3 mg/kg/min up to 150 mg/min	IV: 10-30 min IM: >30 min	>10 h
Phenytoin	20 mg/kg load IV 1 mg/kg/min up to 50 mg/min	15-30 min	>10 h
Phenobarbital	20 mg/kg load IV/IM 1 mg/kg/min up to 100 mg/min	20-30 min	>50 h
Pentobarbital	5-15 mg/kg load IV Maximum rate of 50 mg/min Infusion 1 mg/kg/h	20-30 min	>72 h

Valproic acid	20 mg/kg load IV Infusion 3-6 mg/kg/h	5-15 min	>10 h
Levetiracetam	20-30 mg/kg load IV, then infusion 5 mg/kg/min (maximum 3 g)	60 min	>24 h

IV, intravenous; IO, intraosseous; IM, intramuscular; PR, per rectum; PE, phenytoin sodium equivalents

D. Refractory Status Epilepticus

Refractory SE is defined as continuous seizure activity despite the administration of multiple agents, including benzodiazepines, fosphenytoin or phenytoin, and phenobarbital. Other drugs that are used for refractory SE include: valproic acid, pentobarbital, levetiracetam, and propofol. Often these patients have had continuous (clinical and/or electrographic) seizure activity for several hours. Their care should be a collaborative effort between intensive care specialists and neurologists, and requires standard invasive intensive care monitoring as well as 24-hour availability of an EEG.

Common errors in the management of SE include:

1. *Insufficient initial drug dosages*
2. *Delay in administering a second-line antiepileptic drug*
3. *Inadequate supportive care*

V. ALTERED MENTAL STATUS AND COMA

Case Study

An 18-year-old female young adult collapses while attending a party with friends. On arrival, the emergency medical personnel note that she is weak, unable to stand, and slurring her words. They also note alcohol on her breath. Her pupils are 6 mm, sluggishly reactive. Her blood pressure is 160/110 mm Hg, and she is warm to the touch. Her mental status has continued to deteriorate (GCS score 9), and you are called to assist with intubation and further management.

Detection

– What is the most likely diagnosis?

Intervention

– What indications and techniques should be considered in the airway management of a patient with an altered level of consciousness?

– What medications should be considered for the intubation of a patient with an altered level of consciousness?

Reassessment

– What is the differential diagnosis for a patient with an altered level of consciousness?

– Should any reversal agents be considered?

Effective Communication

– Who needs to be contacted about this patient?

Teamwork

– How are you going to implement the treatment strategy?

The differential diagnosis of coma and altered mental status includes neurologic and non-neurologic diseases and constitutes a true emergency. A systematic approach to these patients increases the likelihood of making the specific diagnosis. Maximal functional recovery depends upon resolution of the primary insult, if possible, and prevention of any secondary injury.

A. Etiologies

Depressed consciousness results from either involvement of both cerebral cortices or the brainstem reticular activating system. This represents an emergency because a prolonged episode potentially limits the likelihood of full recovery. A wide variety of conditions produce alteration of consciousness in children. **Tables 15-6 and 15-7** list the etiologies that produce altered mental status and coma. Trauma, including head trauma, both accidental and nonaccidental, is a common cause of coma in the pediatric patient and is discussed in **Chapters 9 and 11**.

Table 15-6 ETIOLOGIES OF ALTERED MENTAL STATUS AND COMA IN CHILDREN

Ischemic	**Infection**	**Toxins**
Cardiac arrest, cerebrovascular accident, near drowning, shock	Meningitis, encephalitis, brain abscess	Organophosphates, hypoglycemia, drugs of abuse, accidental ingestion
Metabolic	**Trauma**	**Neoplastic**
Diabetic ketoacidosis, hypoglycemia, sodium or osmolar abnormalities, hypercarbia, hyperammonemia	Accidental, nonaccidental	Mass lesions, obstructive hydrocephalus, overproduction of hypoxia, cerebrospinal fluid (choroid plexus)
Inflammatory		
Systemic lupus erythematosus cerebritis		

Table 15-7	COMMON CAUSES OF ALTERED LEVEL OF CONSCIOUSNESS
A	Alcohol
E	Electrolytes, Encephalitis
I	Ingestion, Infection, Insulin
O	Opiates
U	Uremia
T	Trauma
H	Hyper/hypoglycemia, ↑ ↓ temperature, ↑ ↓ blood pressure
I	Intussusception
P	Psychiatric
S	Seizures, Structural

Cerebral hypoxia and hypoxia-ischemia cause a significant proportion of comas in pediatric patients. (See **Chapter 3** for a discussion of global ischemia.) Cerebrovascular accidents (CVAs) producing focal ischemia occur much less commonly in pediatric patients than in adults, but those predisposed to thromboembolic disease may present with focal ischemic CVA. Congenital heart disease places patients at risk for paradoxical emboli and increases their risk for thrombus formation. Thromboembolic CVA may also occur in patients with hemoglobinopathies and hypercoagulable states, including nephrotic syndrome, and protein C and protein S deficiencies. To result in coma, embolic CVAs must involve both cerebral hemispheres (uncommon) and the vertebrobasilar system, which supplies the reticular activating system. Dural sinus thrombosis occurs in severely dehydrated neonates and following infections of the orbit or paranasal sinuses (particularly the sphenoid sinus). These may produce venous infarction of the brainstem and result in coma.

Intoxication may result from either exogenous or endogenous toxins. Hypoglycemia, hyper- or hypocalcemia, hyper- or hypomagnesemia, and hyper- or hypo-osmolality may all produce altered mental status. Any cause of severe acidosis (lactate, inborn error of metabolism) or hyperammonemia (liver failure, inborn error) may also produce altered mental status. Toddlers may accidentally ingest any substance in the home, whereas teenagers may intentionally consume a wide variety of substances. Sedatives (benzodiazepines, barbiturates, and alcohols), tricyclic antidepressants, stimulants (phencyclidine, amphetamines, cocaine), opiates, salicylates, organophosphates, and many other drugs may cause altered mental status (**Chapter 13**).

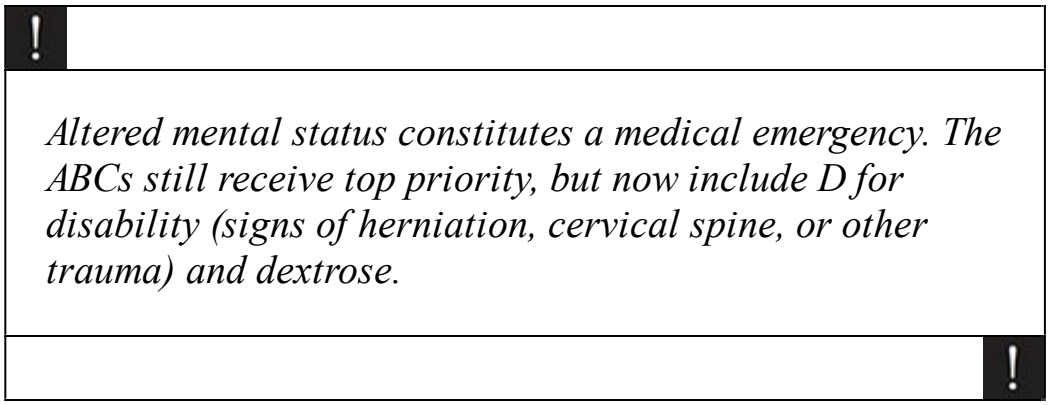
Consciousness may be depressed both during (ictal) and following (postictal) any seizure. Historic evidence for the seizure may be subtle. Prolonged postictal states may occur due to persistence of the cause of the seizure (hypoglycemia, CVA, hyponatremia) or as a consequence of the seizure itself. Neuronal energy demands increase markedly with seizures, and inadequate augmentation of supply may produce ischemic injury.

Meningitis, encephalitis, brain abscess, and subdural empyema may all cause altered mental status. Alterations may result from elevation of ICP, direct inflammation of the reticular activating system,

vasculitis, and subsequent infarctions and seizures. Collagen vascular diseases (systemic lupus erythematosus) may similarly affect the level of consciousness, as may tumors, particularly with acute enlargement (hemorrhage into the tumor) or acute obstructive hydrocephalus.

B. Evaluation

Serial examinations of the level of consciousness are the most important aspect of the neurologic exam. As in all cases, assurance of adequate airway control, respiratory effort, and hemodynamic status is of paramount importance. Because hypoglycemia occurs more commonly in critically ill young children and infants than in adults, the ABCs should be expanded to include D for checking a dextrose (glucose) level and for disability—signs or symptoms of CNS emergencies, such as dilated pupils or the Cushing triad (hypertension, bradycardia, and abnormal respirations). A significant alteration in any of these vital functions may produce altered mental status in a patient. During the resuscitation of comatose patients, 2 key points must be considered. First, if trauma is known or suspected, cervical spine immobilization must be maintained at all times, including during establishment of an artificial airway (**Chapter 2**). Second, fluids for resuscitation should be isotonic or hypertonic to minimize the possibility of cerebral edema and increased ICP.


!
Altered mental status constitutes a medical emergency. The ABCs still receive top priority, but now include D for disability (signs of herniation, cervical spine, or other trauma) and dextrose.
!

Once initial stability has been established and supported, specific evaluation for the cause of the altered/depressed mental status should begin. Many possibilities must be considered in the differential diagnosis of coma or altered mental status in children (**Table 15-6**). Life-threatening causes should be ruled out quickly with a careful history and physical exam, looking for signs of trauma, meningismus, elevated ICP (papilledema), or a toxidrome (**Chapter 13**). Historical information may be limited or difficult to obtain. Specific considerations include the following: Is the patient's mental status worsening/improving? How/where was the patient found, and what was nearby? Were any medications given by family or emergency medical personnel?

Emergent imaging using a CT scan identifies significant intracranial hemorrhage, mass lesions, midline shift, skull fractures, or signs of intracranial hypertension, such as effacement of the sulci or basilar cisterns. The most important consideration prior to a CT scan is whether the patient is stable enough for the procedure. Patients must be adequately resuscitated, and those with compromised airway protective reflexes or respiratory effort should have an airway secured before imaging studies.

A lumbar puncture (LP) permits determination of the opening and closing pressure, cell counts, protein and glucose levels, and culture with Gram stain. As with CT, cardiorespiratory function must

be adequate before LP, particularly in infants and small children whose respiratory function can be severely impaired by positioning for the procedure. There is controversy over whether a CT scan must be performed in all patients prior to LP because it might precipitate herniation in patients with mass lesions or elevated ICP.

!

1. *If cervical spine injury is known or suspected, intubation requires 2 people to provide immobilization. Fiberoptic techniques may be required.*
2. *Resuscitation should proceed with isotonic or hypertonic fluids.*
3. *Rule out life-threatening causes first and treat while the workup proceeds.*

!

However, negative CT scans do not rule out all situations where subsequent herniation may occur after LP (eg, meningitis). Patients with suspected CNS infection and contraindication to LP should be treated with empiric antibiotics (and antiviral agents when appropriate). LP should be performed when deemed safe and may require neurosurgical assistance.

In addition to checking blood glucose, serum electrolytes (including calcium, magnesium, and phosphorus), liver and renal function, arterial blood gases, lactate, and ammonia should also be measured in the patient with altered mental status. A complete blood count may provide evidence of an infectious process or anemia consistent with chronic disease. Urine and serum toxicology screens should be obtained, ideally before the administration of sedatives or analgesics.

C. Therapy

Therapy for coma is directed at the underlying cause, while maintenance of normoxia, normocarbia, and normal perfusion is of paramount importance. Treatment of hypoglycemia with 25% dextrose (2 mL/kg) or 10% dextrose (5 mL/kg) rapidly restores serum glucose levels. Further monitoring and maintenance of normoglycemia (80-150 mg/dL or 4.4-8.3 mmol/L) is prudent. Symptomatic hyponatremia requires an initial, rapid increase in serum sodium to levels that do not produce symptoms; an increase of 5 mEq/L usually will suffice, and 3 to 6 mL/kg of 3% hypertonic saline will provide this increase (**Chapter 8**).

!

1. *When elevated ICP is suspected, adequate airway and circulatory support should precede diagnostic procedures (CT scan, magnetic resonance imaging,*

LP).

2. *Presumptive therapy for intracranial hypertension or CNS infection should be instituted expectantly if diagnostic procedures are delayed.*
3. *Negative findings on a head CT scan do not rule out possible herniation after LP if clinical signs of intracranial hypertension are present.*

VI. INTRACRANIAL HYPERTENSION

Case Study

A 14-year-old boy is brought to the emergency department following a high speed motor vehicle collision. He was an unrestrained passenger in the back seat. His GCS score is 5 on arrival; his trachea is intubated and he is wearing a cervical spine collar. His pulse oximetry reading is 82% and F_{IO_2} is 0.4. You have been called to assist in his management. During your exam, the boy becomes stiff with his upper extremities flexing towards his chest; his blood pressure increases to 170/120 mm Hg, and his heart rate decreases to 55 beats/min.

Detection

– What are the clinical signs and symptoms associated with an ICP elevation?

– What is the Cushing triad?

Intervention

– What are the indications for administration of mannitol and/or hypertonic saline?

Reassessment

– What other therapies could be considered for ICP elevation?

Effective Communication

– Who needs to know about this patient?

Teamwork

– How are you going to monitor this patient?

Although intracranial hypertension (increased ICP) has a number of etiologies, specific management is imperative to promote adequate CPP and CBF.

A. Manifestations

As noted earlier, the Monro-Kellie doctrine states that intracranial hypertension results from any process that increases the volume of either the brain, CSF, or CBV, without a compensatory decrease in one or more of the other components. Increased ICP may produce early findings, such as papilledema and headache. Chronic, slowly progressive increases in ICP are better tolerated than acute increases. Evidence of increased symptoms at night (due to increased $Paco_2$ during sleep), upon awakening (with position changes), or with coughing (increased CBV) may indicate an elevated ICP. Acute increases produce mental status changes and cranial nerve findings much more rapidly. Once intracranial hypertension is suspected, therapy should be instituted immediately and consideration given to ICP monitoring.

B. Monitoring Intracranial Pressure

ICP can be monitored in a number of locations (intraventricular, intraparenchymal, subarachnoid/subdural, and epidural) and by different techniques. The "gold standard" is placement of an intraventricular catheter because it allows drainage of CSF, providing a therapeutic option for increased ICP. The main disadvantages are the difficulty of placement, particularly in patients with compressed ventricles; the increased risk and consequences of infection; and the possibility of malfunction as the ventricles become progressively smaller. The ultimate goal is to recognize elevated ICP. Although ICP >20 mm Hg often triggers interventions, CPP<65 mm Hg (and perhaps between 40 and 50 mm Hg in infants) warrants therapy to raise MAP and/or lower ICP.

!

1. *An ICP >20 mm Hg usually requires specific therapy to lower ICP.*
2. *Maintenance of CPP in the normal range is the primary goal of treatment.*

!

C. Therapy

Assurance of adequate respiratory and circulatory function should receive top priority in all patients, including those with intracranial hypertension. During endotracheal intubation, special consideration should be given to selecting drugs that do not increase ICP and to providing adequate anesthesia to blunt ICP spikes with laryngoscopy. Fever should be aggressively treated to prevent increased cerebral metabolism and CBF. Similarly, seizures should be aggressively treated. Adequate analgesia

and sedation during painful procedures also reduce ICP.

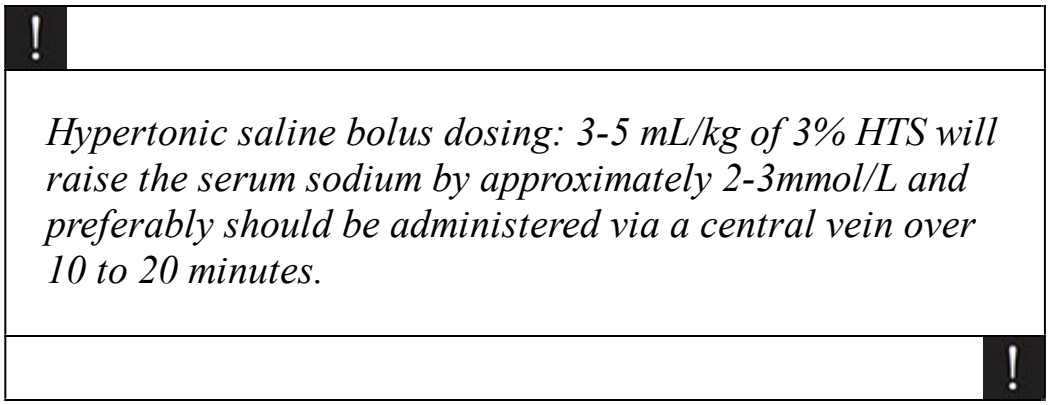
Therapy for elevated ICP begins with the combination of brief induced hyperventilation, sedation, midline neck positioning, and elevation of the head of the bed to 30° (when not contraindicated) to maximize venous drainage. Other therapies aim to reduce the volume of the intracranial contents and/or minimize cerebral metabolic demands.

When intracranial hypertension is suspected, one of the most difficult decisions is determining when to electively sedate and intubate the patient who is otherwise maintaining adequate gas exchange. Although sedation will obscure the clinical neurologic exam, elective intubation is much safer than emergent intubation when apnea or hypoventilation occurs. Once a patient is intubated, however, the need for ICP monitoring increases due to the lack of exam criteria. If a ventricular drain is present, CSF drainage is the next line of therapy. Placement of an extraventricular drain is particularly useful in the initial management of patients with ventricular shunt malfunction, infection, or obstructive hydrocephalus due to neoplasm. Neuromuscular blockade may further lower ICP, even in patients who appear adequately sedated, and has the added benefit of lowering systemic oxygen consumption.

1. *Succinylcholine (without defasciculating non-depolarizer adjunct) may increase ICP.*
2. *Hypoxia and hypercarbia increase ICP.*
3. *Inadequate anesthesia increases ICP with laryngoscopy.*

Osmolar therapy constitutes the next line in medical therapy for intracranial hypertension. Either IV mannitol (0.5 g/kg [range 0.25-1 g/kg]) or hypertonic saline (HTS) may be used to raise serum osmolarity. A 3% HTS solution contains 513 mEq/L of sodium chloride and an osmolarity of 1027 mOsm/L compared with normal saline, which contains 154 mEq/L of sodium chloride and an osmolarity of 308 mOsm/L. Controlled hypovolemia is not appropriate because it places the patient at risk for hypotension and decreased CPP. The diuretic effect of mannitol must be considered in this regard, but diuretics do have a role in the euvolemic or hypervolemic patients with inadequate response to mannitol. Some evidence suggests that higher serum osmolality can be achieved and tolerated with HTS (up to 360 mOsm) than with mannitol (320 mOsm) without increased risk of side effects, but the choice of agent generally depends on rapid availability and the patient's volume status. Sodium distributes in the extracellular fluid space, and 0.6 mEq/kg will raise the serum sodium by 1 mmol/L (**Chapter 8**). Continuous infusions of 3% HTS (0.1-1 mL/kg/h) may be used to maintain the serum sodium (and osmolality) at a level that reduces ICP in patients with traumatic brain injury-induced intracranial hypertension. Importantly, the use of HTS in patients with brain injury and elevated ICP differs dramatically from the management of patients with hyponatremia. In the latter, the rate of increase for sodium correction is limited to <12 mmol per day, because of concerns for inducing central pontine myelinosis. The optimal rate of correction of hyponatremia in a child with severe traumatic brain injury is unclear. Normalization of ICP (and CPP) predominates over a

specific target for the rate of increase in serum osmolality, so the frequency of HTS boluses and rate of infusions will depend on the patient's response as long as serum osmolality remains below 360 mOsm.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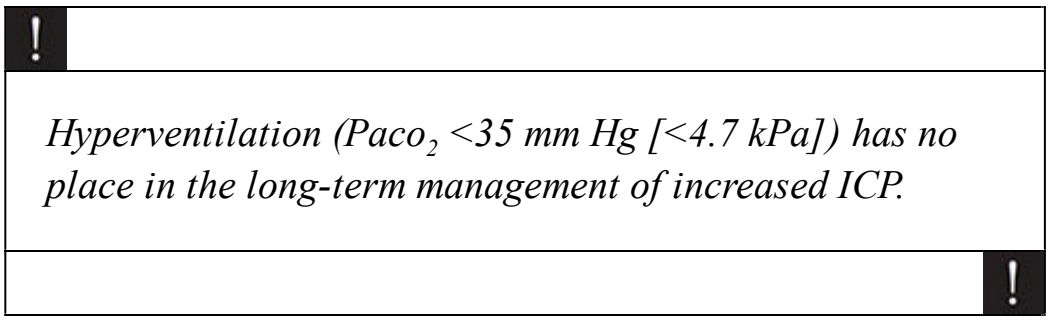
Hypertonic saline bolus dosing: 3-5 mL/kg of 3% HTS will raise the serum sodium by approximately 2-3mmol/L and preferably should be administered via a central vein over 10 to 20 minutes.
!

Other medical therapies may have beneficial effects in limited situations. Corticosteroids (dexamethasone) reduce peritumoral edema but have no beneficial effects in non-neoplastic causes of intracranial hypertension. Careful consideration should be given to gastrointestinal prophylaxis with antacid medications when using corticosteroids because these patients have an increased risk of intestinal ulceration and hemorrhage (Cushing ulcer). High-dose barbiturates inducing pharmacologic coma have proven benefits in reducing ICP, though their effect on outcome remains less clear. In addition, they reduce cerebral oxygen consumption, providing another possible benefit. Barbiturate coma may also be necessary in patients with refractory seizures, which increase ICP.

Two medical therapies deserve special mention. First, aggressive hyperventilation ($Paco_2$ <35 mm Hg [<4.7 kPa]), which may be required briefly for an acute increase in ICP, has no place in the long-term management of intracranial hypertension. As mentioned previously, the reduction in CBF places the brain at risk of ischemia. Also, since the response depends on pH change in the CSF, accommodation occurs with sustained hypocapnia. Note that this also means that a patient with metabolic acidosis (ie, diabetic ketoacidosis) may require a lower $Paco_2$ to maintain a pH of 7.35 to 7.4 and normal CBF until the acidosis is corrected. Second, hypothermia theoretically promotes a number of conditions that reduce ICP. Several studies of traumatic brain injury in animal models and children have documented decreased cerebral oxygen consumption, decreased CBF, reduced inflammation, and altered intracellular signaling with hypothermia. However, clinical studies have demonstrated that outcomes of therapeutic hypothermia (32°C-34°C) and targeted temperature management at 36° C are comparable. Therefore, targeted temperate management may be preferable.



Surgical therapies for elevated ICP include removal of mass lesions (tumor, hematoma) or cranial bones (craniectomy) to increase the compliance of the cranial space. Limited comparative data

support the use of decompressive craniectomy in particular clinical conditions, but it can reduce ICP in patients with medically refractory intracranial hypertension. Another second-line surgical option is the placement of a lumbar drain in patients with slit-like ventricles and increased ICP but with open basilar cisterns. Limited data suggest that this lowers ICP and does not promote herniation.

VII. CEREBROVASCULAR ACCIDENTS

Case Study

An 8-year-old child who is receiving chemotherapy, which includes L-asparaginase and steroids, complains of a new-onset headache; later in the day, the nurse reports that the girl is less responsive with slurring of her speech. You are consulted to assist in her care. She is dysarthric, hemiparetic on the left, with a decreased level of alertness. Her vital signs are significant for mild tachycardia but otherwise normal.

Detection

– What are the common presenting signs and symptoms of a CVA in a pediatric patient?

Intervention

– What laboratory studies should be obtained in a patient with a suspected CVA?

– What neuroimaging studies are required in a patient with a suspected CVA?

Reassessment

– What is the differential diagnosis of suspected CVA?

Effective Communication

– Who should be contacted and where should this patient be managed?

Teamwork

– What is the treatment strategy for a pediatric patient with a CVA?

Although CVAs occur much less commonly in children than in adults, the reported incidence (about 2 cases per 100,000 children each year) closely approximates that of childhood brain tumors. This incidence increases further in specific at-risk populations. Furthermore, CVAs rank among the top 10 causes of death in pediatric patients. Therefore, anyone providing emergency care for children must be able to recognize and manage CVAs. As in adults, therapies must be instituted rapidly to promote maximal functional recovery.

A. Manifestations of Cerebrovascular Accidents

The manifestations of a CVA depend in part on the type of event (hemorrhagic versus thromboembolic) and on the location of the injury. The majority of pediatric CVAs involve the intracranial branches of the carotid artery, specifically the middle cerebral artery. Lesions in this distribution produce hemiparesis, hemianesthesia, hemianopia, and when affecting the dominant hemisphere, aphasia. Because the sensorimotor function for the lower extremities resides on the medial aspect of the cortex, lesions of the anterior cerebral artery affect those functions, as well as emotional control and intellectual function. Lesions of the posterior cerebral artery affect vision or the deep structures of the brainstem, producing motor and sensory defects, pupillary abnormalities, and depressed consciousness. Finally, the vertebrobasilar system (posterior circulation) supplies the lower brainstem and cranial nerves; therefore, deep coma, cranial nerve abnormalities, and cerebellar signs occur with CVAs in that distribution.

B. Hemorrhagic Cerebrovascular Accident

Hemorrhagic stroke, which has a higher mortality rate than ischemic stroke, includes both aneurysms and arteriovenous malformations as primary etiologies, although conversion of initial ischemic events may occur (particularly for venous infarctions). Aneurysms occur either with congenital defects in the muscular layer of arteries or in association with neurocutaneous syndromes and inherited defects of connective-tissue components (ie, Ehlers-Danlos syndrome). Nearly half of aneurysms occur in the posterior circulation, and many occur distally and deep within the brain substance.

Both the increase in ICP and vasospasm contribute to the decreased CBF following aneurysmal bleeding. Blood in the basilar cisterns compromises blood flow to the hypothalamus, resulting in abnormalities of adrenergic tone. This likely contributes to the dysrhythmia and electrocardiogram changes seen with subarachnoid hemorrhage (SAH). Increased sympathetic tone probably produces the neurogenic pulmonary edema seen in these patients.

SAH in children is different than in adults. First, the role of vasospasm in children remains less clear than that in adults. Some studies have suggested similar rates of vasospasm and other studies found a lower rate in children; however, children experience fewer symptoms when vasospasm occurs. Generally, the therapy for vasospasm parallels that for CVAs, with maintenance of adequate CBF being the primary goal. In patients with untreated aneurysms, induced hypertension carries a significant risk of rebleeding and should be avoided. Also, calcium channel blockade probably has a limited role in the treatment of pediatric SAH. Finally, children have a much lower incidence of rebleeding than adults, reducing the need for emergent surgical intervention in the absence of significant mass lesion or intracranial hypertension.

Arteriovenous malformations present most frequently in the second decade of life. Again, most are supratentorial, involving some component of the middle cerebral artery distribution. More than half of patients have intraparenchymal involvement in the hemorrhage, but while SAH may result from extension to the ventricular system or cerebral convexities, blood typically does not involve the basilar cisterns. Therefore, vasospasm is not a concern, although other aspects of therapy for these malformations are similar to those for aneurysmal bleeding. Definitive treatment by surgical excision

or, in some cases, embolization eradicates the 2% to 3% risk of rebleeding per year.

C. Thromboembolic Cerebrovascular Accidents

Thromboembolic disease producing ischemic strokes affects the middle cerebral artery distribution in most cases. Various diseases predispose patients to thromboembolic disease. Cardiac disease, either acquired or congenital, is the most common predisposing factor. Sickle cell disease represents a well-studied predisposition and the only one for which controlled clinical trials have been performed. Infections, including meningitis and paranasal sinus infections, form another large category. Finally, many metabolic and inherited hematologic disorders predispose children to stroke.

Unfortunately, the majority of pediatric patients with ischemic CVAs present late. Intravenous thrombolytic therapy is contraindicated after 3 hours of symptoms. Intra-arterial therapy may be performed up to 8 hours from onset, depending on the arterial territory. Abbreviated diffuse weighted magnetic resonance imaging will help differentiate those patients who may be candidates for thrombolytic therapies. However, thrombolysis in pediatric CVAs remains an infrequent therapy. Most treatment aims to prevent secondary injury by promoting adequate oxygen supply to marginal regions. As mentioned, decreased blood volume, hypotension, and hypocarbia all exacerbate cerebral ischemia. In fact, some authors recommend volume expansion with colloid to induce hemodilution and improve the microvascular rheology. Although this approach remains controversial, prevention of hyperviscosity by maintaining hematocrit in the normal range may be indicated.

Generally speaking, thromboembolic CVAs should remain clinically static after presentation because the embolic event has already occurred. Therefore, deterioration of the neurologic exam should raise suspicion of a secondary insult. Possibilities include hyponatremia, hyper- or hypoglycemia, worsened perfusion, and hemorrhagic conversion. Another possibility is recurrent embolism or extension of clot. Also, edema surrounding the infarcted area typically increases over the first 24 to 48 hours before reaching its peak. Prompt evaluation of each possibility is required. Peak cerebral edema occurs later in patients with secondary insults, such as recurrent emboli, cerebral ischemia secondary to hypotension, or extracellular fluid hypotonicity. If other investigations fail to determine the etiology of the deterioration, consideration should be given to systemic anticoagulation with heparin. Heparinization also may be indicated in patients with documented thrombus and acute thromboembolic CVA if the initial head CT does not detect hemorrhage.

VIII. CEREBROSPINAL FLUID SHUNTS

Case Study

An 8-year-old boy had a ventriculoperitoneal shunt placed following removal of a posterior fossa brain tumor several years ago. He now presents with an acute headache and complains of “just not feeling right.” He has missed school for several days and vomited 3 times before arriving in the

emergency department. His parents report that many children at his school have the flu.

Detection

– What are the common signs and symptoms of ventriculoperitoneal shunt malfunction?

– What are the major complications in patients with shunted hydrocephalus?

Intervention

– What laboratory and neuroimaging studies should be performed?

Reassessment

– What interventions are indicated?

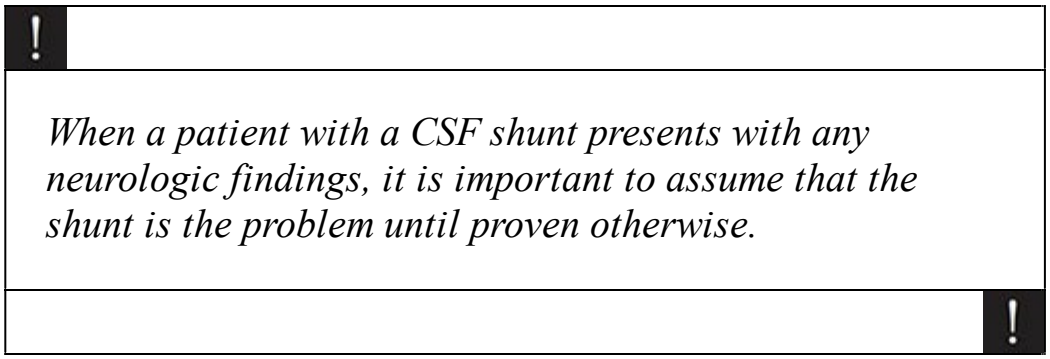
Effective Communication

– Who needs to be contacted about this patient?

Teamwork

– How should the treatment strategy be implemented?

Patients who have shunt obstruction may show signs of increased ICP that include headache, nausea, vomiting, lethargy, and papilledema. It is crucial to involve the neurosurgeon early in the evaluation and management of these patients. Evaluation begins with a thorough history and physical exam. After addressing the ABCs, imaging studies are performed and include a head CT and a shunt series (lateral radiograph of the skull and an anteroposterior view to include the neck, chest, and abdomen to check the shunt position and connections). After these images are reviewed with the neurosurgeon, an attempt to obtain CSF from the shunt is made in a sterile fashion. If this fails or an elevated opening pressure suggests a shunt obstruction, a surgical revision may be necessary. If CSF is obtained, testing includes cell count, culture, Gram stain, and glucose and protein determinations.


!
When a patient with a CSF shunt presents with any neurologic findings, it is important to assume that the shunt is the problem until proven otherwise.
!

Serious complications that may occur in patients with CSF shunts include mechanical failure (proximal or distal obstruction) and infection. The risk of shunt failure is greatest in the first months following placement. Many, if not all, of these children are admitted to the pediatric ICU for observation preceding the surgical repair. Common signs and symptoms include headache, vomiting, nausea, altered mental status, lethargy, and a general feeling of malaise.

Obstruction is the most frequent cause of shunt malfunction, with proximal obstruction occurring more frequently than distal. The proximal end of the catheter may become occluded with choroid plexus, ependymal cells, glial tissue, brain debris, fibrin or blood, or the tip of the catheter may migrate into the brain parenchyma. Distal obstruction may result from kinking or disconnection of the tubing, migration of the catheter outside the peritoneum, intra-abdominal infection, or pseudocyst formation. Regardless of the etiology of the obstruction, occlusion will prevent the drainage of CSF from the ventricular system, which will ultimately lead to a buildup of CSF in the ventricular system and a rise in ICP.

The incidence of CSF shunt infections is between 5% and 8%. Infection may involve the shunt equipment, wound, CSF, or distal site where the shunt drains. Approximately 70% of shunt infections occur within the first 2 months following surgery, and almost 90% will occur in the first 6 months. The organisms most frequently isolated include *Staphylococcus epidermidis* (40%) and *S. aureus* (20%), with the remainder caused by enterococci, streptococci, Gram-negative rods, and yeast. Clinical signs depend on the site of the infection (**Table 15-8**). Wound infections manifest with fever and reddening of the incision or shunt tract, with discharge of pus along the incision as the infection progresses. Patients with ventriculitis and meningitis have fever, headache, irritability, and neck stiffness. The treatment of choice is removal of the shunt hardware, placement of an external ventricular drain, and administration of parenteral antibiotics. Infections of temporary external ventricular drains are managed in the same manner.

Table 15-8 SYMPTOMS OF SHUNT MALFUNCTION

Infants	Toddlers	Adolescents
Fever, vomiting	Fever, vomiting	Fever, vomiting
Head enlargement	Headache	Headache
Tense fontanelle	Irritability and tiredness	Vision problems
Prominent scalp veins	Swelling along shunt track	Irritability and/or tiredness
Swelling along shunt track	Loss of prior abilities (sensory or motor)	Personality change or decline in school performance
Irritability and tiredness	Seizures	Loss of coordination
Downward eye deviation		Difficulty in waking or staying awake

Key Points

Neurologic Emergencies

- In the young child, irritability is an early sign of change in mental status.
- Acute changes in neurologic function, including altered mental status and weakness, require emergent evaluation and treatment to promote maximal recovery.
- Preservation of cerebral blood flow is of paramount importance in any neurologic emergency.

- Hypoxemia, hypercarbia, and hyperthermia should be aggressively prevented in a patient with any neurologic abnormality.
- Intracranial hemorrhage in an infant can cause hemodynamically significant blood loss.
- Hypoglycemia is common in stressed infants and must be corrected promptly.

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MANAGEMENT OF THE CHILD WITH CONGENITAL HEART DISEASE

Objectives

- Identify congenital cardiac defects in neonates who present with low cardiac output.
- Discuss initial management and diagnostic testing of a cyanotic infant.
- Review the evaluation and treatment of children with acyanotic congenital heart disease.
- Understand the most common causes of low cardiac output syndrome in the postoperative pediatric cardiac surgical patient.
- Outline the evaluation and management of low cardiac output syndrome.
- Summarize the most common postoperative arrhythmias.

Case Study

A 4-day-old, full-term male newborn presents with a 1-day history of poor feeding, tachypnea, and pallor. He was born at 39 weeks via normal spontaneous vaginal delivery and was discharged home 39 hours after birth. His parents report that he "acted like a normal baby" until the day before admission, when they noticed he was less active. On arrival to the emergency department, he is lethargic with minimal response to stimuli. His vital signs show a temperature of 35.5°C (95.9°F), heart rate of 180 beats/min, and gasping respirations. His blood pressure could not be obtained by oscillometric technique.

Detection

– What are the most important initial steps in evaluating this neonate?

– What should be included in the differential diagnosis?

– What is the most likely diagnosis?

Intervention

– Which studies and treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– Who needs to be aware of this patient?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, when, and in what order?

I. INTRODUCTION

This chapter will explore the principal issues involved in the care of the pediatric cardiac patient. Although many facets of care are common to all cardiac patients, pediatric cardiac intensive care requires a clear understanding of a patient's native cardiac anatomy and physiology, as well as the circulation changes that follow surgical intervention.

II. NEONATES WITH LOW CARDIAC OUTPUT

A. Hypoplastic Left Heart Syndrome

Hypoplastic left heart syndrome (HLHS) describes a spectrum of congenital heart diseases characterized by varying degrees of hypoplasia of the left-sided heart structures. The anatomic variants of the disease are distinguished by the status of the left heart valves (eg, aortic atresia, mitral atresia). The incidence in the United States is 2.4 cases per 10,000 live births. HLHS is characterized by a tiny left ventricle (LV), incapable of supporting the systemic circulation, and underdevelopment of the mitral and aortic valves. The central anatomic feature in the most common form is aortic valve atresia with hypoplasia of the ascending aorta and aortic arch with a coarctation of the aorta.

The LV is a nonfunctional structure in infants with HLHS, with both systemic and pulmonary venous return mixing in the right atrium and the right ventricle (RV), supplying both the systemic and pulmonary circulation in a parallel fashion. Blood flows from the RV via the pulmonary artery (PA), through the ductus arteriosus (right to left) and retrograde to the transverse then ascending aortic arch, ultimately to provide coronary perfusion. Blood flow to the lower body is from the RV through the ductus arteriosus (right to left) and then to the descending aorta. Closure of the ductus arteriosus leads to markedly decreased systemic and coronary perfusion thus the clinical feature of the "pulseless

cardiac disease in all 4 extremities". Survival beyond birth depends on the persistent patency of the ductus arteriosus to maintain the systemic and coronary circulation. Without intervention, HLHS is fatal within the first few days after birth. Neonates typically present with mild cyanosis, tachypnea, and signs of shock after the ductus arteriosus begins to close. An arterial blood gas measurement will show a lactic acidosis due to decreased systemic perfusion and hypoxemia, typically with a Pao_2 <100 mm Hg (<13.3 kPa).

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Any infant with symptoms of shock and severely diminished or absent pulses in the first weeks after birth must be suspected of having a ductal-dependent lesion like HLHS.

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Infants with HLHS who have a widely patent ductus arteriosus will present with normal to slightly decreased peripheral pulses and mild or no metabolic acidosis. Auscultation usually shows a systolic ejection murmur and a single second heart sound (S2). Infants who have a marked imbalance between pulmonary and systemic blood flow (Qp:Qs) due to closure of the ductus arteriosus will present in cardiogenic shock. The degree of cyanosis is determined by the size of the interatrial communication, with extreme cyanosis in those with a very restricted atrial septal defect or intact atrial septum that restricts mixing of oxygenated and deoxygenated blood.

The physical findings of mild cyanosis, respiratory distress, and impending cardiovascular collapse are nonspecific and do not help to distinguish this ductal-dependent cardiac lesion from others that present in the neonatal period. The electrocardiogram shows right ventricular forces predominantly, as in normal neonates, but without the presence of LV forces. Cardiomegaly and pulmonary edema are present on the chest radiograph. An echocardiogram defines the anatomic details, confirms the diagnosis, and should be performed urgently.

Timely and appropriate initial medical management can be lifesaving. Prostaglandin E_1 (PGE_1) infusion should be initiated rapidly to reopen the ductus arteriosus and restore perfusion to the descending aorta and, through retrograde flow, to the ascending aorta and coronary circulation. The usual starting dose of PGE_1 is 0.05 µg/kg/min (range 0.025-0.1 µg/kg/min). The major side effects of PGE_1 include apnea and peripheral vasodilatation, so the clinician must be prepared to provide mechanical ventilator support and administer additional fluids and/or inotropic agents if needed. Infants who are diagnosed prenatally or who present with stable hemodynamics may benefit from spontaneous breathing. However, the infant who presents in cardiogenic shock with critically low oxygen delivery requires control of the airway and hemodynamic support with intubation and mechanical ventilation.

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For children with HLHS, PGE_1 infusion is the most

important component of the initial therapy and resuscitation. Any newborn with unexplained shock should be started on PGE_1 infusion until a ductal-dependent lesion has been ruled out.

Once the ductus arteriosus has reopened and distal perfusion is restored, maintaining a favorable balance between the systemic and pulmonary circulations is critical for preserving distal organ perfusion while maintaining adequate pulmonary blood flow. Hyperventilation and hyperoxia will both lead to preferential pulmonary blood flow through the ductus arteriosus at the expense of systemic blood flow. The fraction of inspired oxygen should be rapidly weaned to the lowest level (as low as 0.21) to reach a targeted oxygen saturation level between 75% and 85% on pulse oximetry. Judicious fluid administration (5-10 mL/kg of 0.9% saline or 5% albumin) and inotropic support (3-5 μg/kg/min dopamine or 0.01-0.05 μg/kg/min epinephrine) may be necessary in certain cases to support cardiac output.

The specifics of surgical palliation for HLHS may vary, but in most centers, a staged surgical approach is undertaken during the first week after birth to create a neo-aorta and provide adequate pulmonary circulation (**Table 16-1**). The three goals of stage I reconstruction (ie, Norwood operation) for HLHS are: 1) to provide systemic perfusion independent of the ductus arteriosus; 2) to preserve ventricular function, preventing volume and pressure overload; and 3) to allow for balanced pulmonary and systemic blood flows. The procedures consist of: 1) anastomosing the main pulmonary trunk to the underside of the diminutive aortic arch with arch reconstruction and coarctectomy (Damus-Kaye-Stansel procedure); 2) creation of a systemic-to-pulmonary artery shunt, either with an innominate-to-pulmonary artery graft (modified Blalock-Taussig shunt) or via an RV to pulmonary artery conduit (Sano shunt) to provide a source of pulmonary blood flow; and 3) atrial septectomy to allow for adequate atrial level mixing of oxygenated and deoxygenated blood while avoiding pulmonary venous hypertension. Recently a hybrid approach has been developed to accomplish similar tasks but in the interventional cardiac catheterization laboratory in concert with the cardiac surgeons. A stent is placed in the patent ductus arteriosus to permanently open the vessel, and an atrial septostomy is performed by the cardiologists; this is followed by placement of bilateral PA bands via a median sternotomy by the cardiac surgeons. Either of the first-stage procedures (Norwood or hybrid) will then lead to a cavopulmonary anastomosis, connecting the superior vena cava to the right pulmonary artery (bidirectional Glenn anastomosis), when the patient is between 3 and 6 months of age. The third stage (Fontan procedure) incorporates the inferior vena cava into the pulmonary circulation and is performed at approximately 2-4 years of age. Successful completion of the third stage allows the entire systemic venous return to completely bypass the right side of the heart and flow directly into the pulmonary arteries. Long-term survival with good neurologic outcome is attainable at centers that specialize in complex congenital cardiac surgery.

Maintaining balance between pulmonary and systemic

circulation is critical to maintaining distal organ perfusion. A pulmonary to systemic blood flow (Qp:Qs) of 1 is ideal. Oxygenation goals should aim for saturation between 75% and 85%.

Table 16-1	STAGED SURGICAL REPAIR OF HYPOPLASTIC LEFT HEART SYNDROME	
Procedure	**Age of Patient**	**Actions Performed**
Stage I (Norwood procedure)	First weeks after birth	Patent ductus arteriosus is ligated. Main PA is separated from branch PAs and used to create a neo-aorta and augmented aortic arch using homografts. Pulmonary blood flow is reestablished via either a modified Blalock-Taussig shunt (innominate or subclavian artery to ipsilateral PA) or Sano shunt (RV to PA conduit). Atrial septectomy is performed. Regulation of pulmonary blood flow with a shunt of fixed diameter and length, coupled with nonrestrictive interatrial communication via the atrial septectomy, enables balanced pulmonary blood flow with near-normal development of the pulmonary vasculature while avoiding excessive ventricular volume overload.
Stage II (modified bidirectional Glenn anastomosis)	3-6 months	Takedown of modified Blalock-Taussig shunt or RV to PA conduit (Sano shunt) is performed. Distal superior vena cava is sewn into the top of the right PA (end-to-side anastomosis).
Stage III (Modified Fontan procedure)	2-4 years	Inferior vena cava is connected to the underside of the branch PA via an extracardiac procedure conduit.

PA, pulmonary artery; RV, right ventricle

A third option in the surgical management of HLHS is to place the infant on the cardiac transplantation list. Because organ availability for neonates is limited, waiting times are prolonged, and stage I reconstruction at experienced centers carries acceptable (if not very good) results, primary heart transplantation for HLHS is rarely offered. However, it is considered in neonates with severe RV dysfunction and/or severe tricuspid regurgitation.

B. Aortic Stenosis

Abnormalities of the aortic valve account for about 80% of the left ventricular outflow tract obstruction lesions. In the United States, aortic stenosis occurs in 3% of children with congenital heart disease (CHD). The commissures of the aortic valve are fused, resulting in a thickened, domed

valve with a stenotic orifice. The presenting symptoms and findings on physical examination are variable and relate to the degree of obstruction. Patients have a systolic ejection murmur loudest at the upper right sternal border with radiation into the neck. Often a click is audible.

Patients with critical aortic stenosis have markedly decreased systemic perfusion, resulting in cardiogenic shock. They typically have a history of poor oral intake and decreased urine output. On examination, these infants are tachypneic and lethargic, with cold extremities and diminished peripheral pulses throughout. Chest radiographs reveal cardiomegaly and pulmonary edema, and laboratory results reveal lactic acidosis.

Neonates with cardiovascular collapse require emergent treatment with PGE_1 infusion to reestablish ductal-dependent systemic flow. Intubation, mechanical ventilation, and inotropic support are needed in these critically ill patients.

Balloon valvuloplasty of the aortic valve is performed in the cardiac catheterization laboratory and is often successful in treating critical aortic stenosis. Complications include severe aortic regurgitation at the time of the procedure and restenosis afterward. Surgical valve replacement with a prosthetic mechanical valve or the patient's native pulmonary valve (Ross procedure) is recommended for patients in whom the valve annulus is very small or after failed valvuloplasty. Enlargement of the left ventricular outflow tract with a patch (Konno procedure) may be required at valve replacement to make room for the valve in the left ventricular outflow tract.

Beyond the neonatal period, aortic stenosis has a very different course. These patients have lower degrees of outflow obstruction with adequate distal flow. They are mostly asymptomatic and present with a loud systolic murmur. In general, intervention is indicated when the pressure gradient across the aortic valve is >50 mm Hg or when patients are symptomatic (eg, chest pain, exercise intolerance, syncope).

C. Coarctation of the Aorta

Coarctation of the aorta (CoA), which occurs in 5% of children with CHD, is a narrowing of the descending aorta that usually occurs opposite to the ductus arteriosus. The symptoms vary depending on the degree of obstruction. Infants with critical CoA present with cardiogenic shock in the neonatal period, while those with mild disease often present with hypertension or an asymptomatic murmur later in childhood.

Neonates with critical CoA are fussy, have difficulty feeding, and become lethargic over time. On exam, femoral pulses are decreased while pulses in the right upper extremity are normal or bounding. The left arm pulse is variably involved with the coarctation. If the diagnosis is missed, these patients progress to cardiogenic shock with respiratory failure and/or congestive heart failure.

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Infants with critical coarctation present with shock in the first week after birth, after spontaneous closure of the ductus arteriosus. Left ventricular ejection fraction

decreases acutely in response to the increased afterload, leading to elevated ventricular wall tension, decreased myocardial perfusion pressure, and the onset of myocardial ischemia.

Most neonates with CoA undergo surgical repair, although balloon angioplasty or stent implantation can be performed in selected patients at some centers. Many older children with CoA have postoperative hypertension that is usually responsive to β-receptor blockade agents or angiotensin-converting enzyme inhibitors. In the immediate postoperative period, esmolol (50-250 μg/kg/min) has the advantage of a very short half-life, which allows for easy titration. Patients normally transition to oral therapy with either atenolol (0.5-2 mg/kg/day) or enalapril (0.05-0.25 mg/kg given twice daily); the dose is titrated as necessary (maximum dose 5 mg) to maintain a normal blood pressure.

PGE_1 infusion (initial dose 0.05-0.1 μg/kg/min) is lifesaving for neonates with critical CoA who are in shock.

D. Myocarditis and Cardiomyopathies

Myocarditis and cardiomyopathies may present in the neonatal period but are much more common at older ages. Lesions are characterized by ventricular dysfunction. Patients typically present with signs and symptoms of congestive heart failure (**Tables 16-2** and **16-3**) but may also exhibit cardiogenic shock.

Table 16-2 COMMON SYMPTOMS OF HEART FAILURE

Infants	Children
Tachypnea	Failure to thrive
Diaphoresis	Decreased energy
Feeding intolerance	Exercise intolerance
Decreased urine output	Frequent respiratory infections
Failure to thrive	Chest pain

Table 16-3 COMMON PHYSICAL FINDINGS OF CONGESTIVE HEART FAILURE

Tachycardia	Tachypnea
Displaced and dampened apical impulse	Failure to thrive
Gallop rhythm (S3 and S4)	Pulmonary rales
Hepatomegaly	Peripheral edema

Case Study

An 8-year-old girl presents with viral upper respiratory tract symptoms of 1 week's duration, low-grade fever, and new onset of anorexia, tachypnea, vomiting, and diffuse abdominal pain. On arrival to the emergency department, she appears ill and fatigued. Her initial vital signs are: temperature, 37.7°C (99.8°F); heart rate, 140 beats/min; respiratory rate, 36 breaths/min; and blood pressure, 70/30 mm Hg. A chest radiograph shows massive cardiomegaly (**Figure 16-1**)

Figure 16-1. Chest Radiograph Showing Massive Cardiomegaly

Detection

– What are the most important initial steps in evaluating this child?

– What should be included in the differential diagnosis?

– What is the most likely diagnosis?

Intervention

– Which studies and treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– Who needs to be aware of this patient?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, when, and in what order?

1. Cardiomyopathies

Cardiomyopathies may present in the neonatal period but are more common in older children. Cardiomyopathies can be classified as *dilated, hypertrophic*, or *restrictive* and categorized according to the etiology (eg, infectious, drug induced, genetic).

Patients with *dilated cardiomyopathy* (DCM) present with varying degrees of heart failure. In infants, this can be manifested by poor feeding and failure to thrive; in older children and adolescents, the clinical signs of congestive heart failure are often present; these include rales, tachypnea, hepatomegaly, and poor peripheral perfusion. Many children with DCM present with abdominal pain, which is related to an enlarged liver and intestinal hypoperfusion. The cardiac exam reveals tachycardia, a gallop, and often a heart murmur due to atrioventricular valve regurgitation. Echocardiography allows quantification of ventricular volume and function and can rule out many anatomic causes of ventricular dysfunction (ventricular outflow tract obstruction, atrioventricular valve regurgitation) or coronary anomalies (ie, anomalous left coronary artery arising from the pulmonary artery). Cardiac magnetic resonance imaging (MRI) is a helpful tool for evaluating these patients because it provides ventricular volume and ejection fraction quantification, as well as some specific myocardial abnormalities (eg, left ventricular noncompaction cardiomyopathy, arrhythmogenic RV cardiomyopathy, obstructive hypertrophic cardiomyopathy).

The initial treatment of DCM aims to manage heart failure and prevent and treat common complications like thromboembolism and arrhythmias. Acute short-term management is directed toward improving ventricular function by increasing contractility, optimizing preload, and decreasing afterload. The use of an inodilator (eg, milrinone, 0.25-0.75 μg/kg/min) is often indicated. This is often combined with low-dose epinephrine to provide synergistic inotropic support. A high-dose catecholamine infusion should be avoided as this can result in marked tachycardia, increased systemic vascular resistance, and thus increased afterload to the impaired ventricle. In the intermediate term, high-dose catecholamines can induce apoptosis, resulting in catecholamine-induced cardiomyopathy. Reduction of right and left end-diastolic pressure is accomplished with adequate diuresis, often induced with furosemide.

Endotracheal intubation and positive pressure ventilation can decrease ventricular transmural pressure and the work of breathing, which will benefit greatly by decreasing oxygen consumption and increasing oxygen delivery. However, endotracheal intubation is not without risk, and it should be performed by experienced personnel with a clear understanding of the patient's physiology and condition, particularly with regards to the anesthetic agents used. Fentanyl, etomidate, and ketamine are agents commonly used, each with its own risk and benefit. Fentanyl (1-2 μg/kg) is a synthetic centrally acting opioid agonist that has a rapid onset of action and short duration of effect. It is

favored by some experts for rapid sequence intubation in children with cardiogenic shock or shock with suspected catecholamine depletion. Etomidate (0.3 mg/kg) is an ultra-short imidazole derivative that produces adequate sedation and induction for rapid sequence intubation in these patients without significant hemodynamic effects; however, despite its safe hemodynamic profile, etomidate has been associated with induction of adrenal insufficiency and worse outcomes in septic patients. Ketamine (0.5-1 mg/kg) is a safe and valuable alternative to etomidate, but it can exacerbate myocardial depression in catecholamine-depleted patients.

Anticoagulation is indicated in patients with severe ventricular dysfunction to prevent thromboembolic complications. Low-molecular-weight heparin has emerged as a preferred choice for the acute management of anticoagulation. Optimization of hemoglobin levels continues to be an important facet of critical care, although a more restrictive approach has emerged in recent years. Mechanical circulatory support (extracorporeal life support or ventricular assist devices) should be considered as a bridge to recovery or heart transplantation in severely affected patients who present with severe heart failure refractory to maximal medical therapy.

Hypertrophic cardiomyopathy (HCM) can present early in infancy or in older children and adolescents. It can involve one or both ventricles and is further classified as obstructive or nonobstructive. Infants and young children often present with increased work of breathing and failure to thrive. Angina in young infants is manifested by crying or agitation out of proportion to other infants and less responsive to soothing measures. Older children usually complain of dyspnea, angina, or syncope. Sudden death during athletics is possible. Echocardiography, cardiac MRI, and electrocardiogram are all tools that aid in the diagnosis. Medical management requires lusitropic agents (usually β-blockers) that can help to relax the myocardium during diastole and decrease outflow tract obstruction while decreasing heart rate; this further diminishes myocardial oxygen consumption. β-blockers also decrease circulating catecholamines, thus decreasing surge which could lead to sudden death. Calcium channel blockers have been shown to improve diastolic function. At-risk patients have automated implantable cardioverter defibrillators placed to prevent sudden death.

Restrictive cardiomyopathy (RCM), the least common form of cardiomyopathy (2%-5% of pediatric cardiomyopathy patients), is characterized by low cardiac output with ventricular diastolic dysfunction evident by high diastolic filling pressures and marked atrial dilatation in the context of normal systolic function and ventricular thickness. Pediatric patients with this form have a rapidly fatal course (50% fatal within 2 years of diagnosis), and arrhythmias are common and often fatal. Ventricular tachyarrhythmias and high-grade atrioventricular block have been documented in children with restrictive cardiomyopathy and are presumably the terminal events.

Case Study

A 14-year-old boy is admitted with fever, cough, and vague diffuse abdominal pain. His parents report that he has asthma and was diagnosed with a flu-like illness 5 days ago. His vitals signs reveal: temperature, 36°C (96.8°F); heart rate, 140 beats/min; blood pressure, 80/60 mm Hg; and respiratory rate, 30 breaths/min. Physical exam shows a pale, ill-appearing adolescent with cool extremities and weak pulses.

Detection

– What are the most important initial steps in evaluating this patient?

– What should be included in the differential diagnosis?

– What is the most likely diagnosis?

Intervention

– Which studies and treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– Who needs to be aware of this patient?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

2. Myocarditis

The clinical presentation of myocarditis is variable. Patients can have different degrees of severity, from subclinical disease to cardiogenic shock, arrhythmias, and sudden death. The most common etiology is viral (**Table 16-4**), and patients usually present with a history of a recent upper respiratory infection or gastrointestinal illness. Patients with *acute myocarditis* usually exhibit signs of heart failure, but nonspecific signs and symptoms such as gastrointestinal symptoms (abdominal pain, nausea, vomiting) may be the most predominant symptom and can lead to an incorrect diagnosis. Arrhythmias are common at presentation and during the course of the disease. Myocarditis should be suspected in a patient with acute onset heart failure and no structural heart disease, especially after a viral illness. Initial tests include electrocardiogram, cardiac biomarkers (cardiac troponin I, β-natriuretic peptide), chest radiograph, and echocardiography. Endomyocardial biopsy has been considered the "gold standard" in confirming a clinical diagnosis of myocarditis, although this is now rarely performed given its low sensitivity and the risks involved with the procedure. Cardiac MRI has been increasingly used to aid with the diagnosis of myocarditis; consensus diagnostic MRI criteria have been published. Studies in adults have found a diagnostic accuracy close to 80%. Larger pediatric studies are needed to determine the validity of cardiac MRI in children with myocarditis. Rapid viral screening (immunofluorescence, polymerase chain reaction), viral culture, and acute versus convalescent antibody titers are diagnostic.

Table 16-4	COMMON VIRAL AGENTS CAUSING MYOCARDITIS IN CHILDREN

Coxsackievirus types A and B (especially type B)
Adenovirus (most commonly types 2 and 5)
Epstein-Barr virus
Cytomegalovirus
Herpes virus
Human immunodeficiency virus
Influenza
Parainfluenza
Measles
Mumps
Parvovirus B19

A subset of critically ill patients present with acute *fulminant myocarditis*. These patients usually have fever and a 2- or 3-day history of a viral illness; they are in profound shock with signs of markedly decreased cardiac output, hypotension, poor pulses, decreased perfusion, severe lactic acidosis, and multiorgan system failure. Malignant tachyarrhythmias are common in these cases. Fulminant myocarditis has a high mortality rate if not treated aggressively.

During the acute phase, management includes rapid recognition and maximum medical therapy aiming to improve cardiac output as in DCM. Myocarditis patients are treated early with immunomodulatory therapy with IV immunoglobulin. Typical treatment schedules include 2 g/kg (as a 5% or 10% solution) over 1 or 2 days, depending on the patient's ability to handle the volume infused. Aggressive treatment of tachyarrhythmias is necessary as they are often fatal if left untreated. Amiodarone (5 mg/kg load with continuous infusion for malignant tachyarrhythmias at 5-10 μg/kg/min) is used, but extreme caution is needed in administering the loading dose as the α, β, and calcium channel blocking properties depress cardiac function further and can be fatal. When possible, infusing the loading dose over 1 hour is preferred. Synchronized biphasic direct current cardioversion should be available and used to treat ventricular tachycardia; asynchronous cardioversion is used for ventricular fibrillation. Meticulous electrolyte (especially potassium, magnesium, calcium, and phosphorus) monitoring and correction of any abnormalities must be undertaken. Patients refractory to these therapies require mechanical circulatory support with extracorporeal life support or a ventricular assist device (typically a biventricular device), and early institution is key to avoiding end-organ failure, including neurologic injury. The choice between method of circulatory support depends on patient size, availability of the individual devices, and center expertise. Finally, outcomes in acute fulminant myocarditis are improved when a dilated and hypertensive left atrium is decompressed with either a balloon septostomy of the atrial septum or a puncture of the atrial septum with subsequent balloon dilation or blade incision. The former might be performed at the bedside, whereas the latter requires transfer to the cardiac catheterization lab.

3. Pericarditis/Pericardial Effusion/Cardiac Tamponade

Pericarditis is an inflammatory process involving the pericardium. The etiology of pericarditis includes infectious and non-infectious causes. Patients with pericarditis present with chest pain and fever. Those with a large pericardial effusion present with dyspnea and shortness of breath. During the initial accumulation of a pericardial effusion, cardiac output is not usually compromised;

however, as the effusion increases, signs of decreased cardiac output become evident. The physical exam usually reveals a pericardial rub (small effusions) or muffled heart sounds (large effusions). Acute cardiac tamponade is a life-threatening emergency. It is characterized by compression of all cardiac chambers secondary to increased pericardial pressure. Patients present with decreased peripheral perfusion, distended jugular veins, and pulsus paradoxus manifested as a large decrease in systolic blood pressure during inspiration (>10 mm Hg). Pulsus paradoxus indicates critical cardiac compression by pericardial contents with markedly decreased intracavitary volumes; the cardiac output is further reduced during inspiration, leading to a drop in the systolic blood pressure.

The electrocardiogram can reveal low-voltage complexes and elevation of ST segments with the axis of the heart changing beat to beat. A chest radiograph shows an enlarged cardiac silhouette with signs of pulmonary edema. An echocardiogram demonstrates the presence and size of the pericardial effusion and is diagnostic. Tamponade with right atrial and right ventricular collapse can be easily seen, particularly during inspiration. Doppler interrogation of the inflows to both the tricuspid and mitral valves reveals significant decreases in the peak velocity during inspiration.

Treatment of cardiac tamponade includes rapid fluid resuscitation to increase ventricular filling. Diuretics are contraindicated as hypovolemia can severely worsen ventricular filling. Pericardiocentesis should be performed as quickly as possible. A pigtail catheter drain (5F for neonates, 7F-8F for children, 8F-10F for adolescents) is typically sutured in place and gentle suction applied (-10 cm H_2O) for continued evacuation of pericardial fluid. This lifesaving procedure is usually performed in the ICU under echocardiographic guidance with judicious use of local (1% lidocaine) and IV analgesia (opioids). Particular care is needed to avoid decreasing the systemic blood pressure to a critically low level. Ketamine can be a useful agent for sedation and analgesia when tamponade is present and the patient is already hypotensive.

The causes of low cardiac output in newborns are listed in **Table 16-5**, and a summary of the initial evaluation and treatment of low cardiac output is presented in **Table 16-6**.

Table 16-5	CAUSES OF LOW CARDIAC OUTPUT IN NEWBORNS
Coarctation of the aorta	Myocarditis
Hypoplastic left heart syndrome	Pericardial tamponade
Critical aortic stenosis	Pneumothorax
Interrupted aortic arch	Hydrops fetalis
Arrhythmia (tachycardic or bradycardic)	Sepsis (decompensated, late-stage)
Cardiomyopathy (hypertrophic from maternal diabetes)	

Table 16-6	INITIAL EVALUATION AND TREATMENT OF NEWBORNS WITH LOW CARDIAC OUTPUT
Initial Evaluation	**Initial Treatment**
Blood culture	Intravenous fluid resuscitation: 5-10 mL/kg bolus of normal saline or 5% albumin or packed red blood cells (if anemic). Repeat if clinically indicated.
Arterial blood gas	Inotropic agents

Central venous oxygen saturation	Prostaglandin E_1 (for ductal-dependent lesions): range 0.025-0.1 µg/kg/min IV
Lactate	Treatment of underlying arrhythmia
Chest radiograph	IV immune globulin for myocarditis
Electrocardiogram	Antibiotics
Echocardiogram	
	NOTE: Oxygen can be detrimental in hypoplastic left heart syndrome.

III. CYANOTIC CONGENITAL HEART DISEASE

Case Study

A 4-kg male neonate is delivered by normal spontaneous vaginal delivery 10 hours after spontaneous rupture of membranes to a 22-year-old mother with an uncomplicated pregnancy. Delivery was unremarkable with reassuring fetal heart tones immediately before delivery. The cord pH was 7.3. His initial Apgar score is 8/8 (-2 color), and he is noted to be deeply cyanotic. Pulse oximetry readings are 75% (preductal, right arm) and 55% (postductal, right leg). He is tachypneic and vigorous initially, but he becomes progressively less responsive (10-min Apgar score of 6).

Detection

– What are the most important initial steps in evaluating this newborn?

– What should be included in the differential diagnosis?

– What is the most likely diagnosis?

Intervention

– Which studies and treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– Who needs to be aware of this patient?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, when, and in what order?

Both central cyanosis and acrocyanosis may be seen in newborns. Acrocyanosis involves the hands and the feet and does not represent true arterial hypoxemia. Central cyanosis—present throughout the body, but more evident in the mucous membranes, lips, and tongue—represents true hypoxemia. For central cyanosis to be recognized clinically, >5 g/dL of hemoglobin must be deoxygenated.

A. Cyanotic Congenital Heart Disease with Decreased Pulmonary Blood Flow

Patients with cyanotic CHD and decreased pulmonary blood flow have obstructed pulmonary flow and right-to-left shunting of blood at either the atrial or ventricular level. Neonates with these defects are usually cyanotic in proportion to the degree of pulmonary stenosis, but they are rarely dyspneic as there is an absence of pulmonary overcirculation or congestive heart failure.

1. Tetralogy of Fallot

Tetralogy of Fallot (TOF) is the most common form of cyanotic CHD, occurring in 7% of US children with congenital heart disease. The tetralogy consists of:

- Pulmonary stenosis (subvalvular, valvular, and/or supravalvular)
- Ventricular septal defect (VSD)
- Aorta overriding the ventricular septum
- Right ventricular hypertrophy

From a pathophysiologic standpoint, the dominant features of TOF are the VSD and the degree of right ventricular outflow tract obstruction (RVOTO). Patients with mild RVOTO have signs of overcirculation due to a left-to-right shunt through the VSD. These patients have normal saturations ("pink TOF") and signs of congestive heart failure. However, in patients with severe RVOTO, significant right-to-left shunt occurs at the VSD level. In these patients, a patent ductus arteriosus (PDA) maintains normal pulmonary blood flow in the immediate postnatal period. The degree of cyanosis depends on the severity of the pulmonary stenosis and worsens as the PDA closes. A hypercyanotic spell, or tetralogy spell ("tet spell"), is a characteristic sequence of clinical events that begins with irritability and hyperpnea, followed by a prolonged period of profound cyanosis and leading to syncope due to near-complete RVOTO. Tetralogy spells are more common in the morning after a prolonged overnight fast and at 2 months of age as the infant reaches the physiologic nadir for hemoglobin. β-blockers may be prescribed for infants with TOF to lessen the occurrence of spells.

Characteristic sequence of hypercyanotic spell:

- *Irritability and hyperpnea*
- *Intense cyanosis*
- *Syncope*

Treatment:

- *Place the child in knee-chest position.*
- *Provide maximal supplemental oxygen.*
- *If necessary, give morphine, 0.1 mg/kg intramuscularly (if no IV access is present)*
- *Phenylephrine, 5-20 µg/kg/dose*
- *Correction of acidosis*
- *Isotonic fluid bolus*

Prompt medical treatment is lifesaving in children with hypercyanotic spells. Initially, attempts should be made to calm the child, who then should be placed in a knee-to-chest position. Maximal supplemental oxygen by face mask should be provided. If the spell continues, morphine (0.1 mg/kg) may be given intramuscularly; if it persists, more aggressive therapy is initiated, including rapid placement of an IV catheter and administration of isotonic IV fluid (20 mL/kg of 0.9% saline) and sodium bicarbonate (1-2 mEq/kg). Administration of a β-blocker may be needed to relax the RVOTO and increase pulmonary blood flow. In severe cases that do not respond to these measures, intubation and administration of an α-adrenergic agonist (phenylephrine, 5-20 µg/kg/dose) may be needed to increase systemic vascular resistance (SVR) and decrease right-to-left shunting at the VSD. Anemia (hemoglobin <10 g/dL), when present, often precipitates a hypercyanotic spell, and blood transfusion may be required.

Surgical repair of TOF is indicated for any child with hypercyanotic spells or increasing cyanosis (oxygen saturation <75%). In the past, symptomatic TOF was repaired utilizing a staged approach, with placement of a modified Blalock-Taussig shunt (a polytetrafluoroethylene tube connection between the innominate and pulmonary arteries) in early infancy as a palliative measure. Definitive repair—pulmonary valvotomy, resection of right ventricular infundibular muscle bundles, and VSD closure—was then performed at 3 or 4 years of age. With advances in medical and surgical management of younger infants with CHD, elective repair is now performed as a single surgical procedure at 4 to 6 months of age. Debate continues on the optimum surgical course for ductal-dependent lesions in neonates with TOF, with some centers advocating complete repair in the neonatal period. Results are promising, although complications and death occur more often in these high-risk patients compared to those undergoing the 2-stage approach (palliation, followed by complete repair outside the neonatal period).

2. Tricuspid Atresia

Tricuspid atresia with normally related great arteries is a rare form of CHD in which the tricuspid valve orifice is not patent. A patent foramen ovale or atrial septal defect must be present to allow systemic venous blood to shunt from right to left. The RV and pulmonary outflow tract are typically

hypoplastic unless a large VSD is present. Patients with tricuspid atresia and normally related great arteries present in the immediate neonatal period with cyanosis (oxygen saturation ~70%). Initial medical therapy includes the initiation of PGE_1 infusion (0.05 μg/kg/min) to maintain the patency of the ductus arteriosus. Palliative surgery is performed in the first week after birth and consists of placement of a modified Blalock-Taussig shunt. A bidirectional Glenn procedure (cavopulmonary anastomosis connecting the superior vena cava to the right pulmonary artery) is performed at 4 to 6 months in a staged approach, followed by the modified Fontan procedure at approximately 2 years of age. The modified Fontan procedure allows systemic venous blood to completely bypass the right side of the heart by directing the inferior vena cava through an extracardiac conduit into the pulmonary arteries. This staged surgical repair is summarized in **Table 16-1**.

3. Pulmonary Stenosis

Pulmonary stenosis occurs in 9% of children with heart disease, most of whom present with a harsh systolic ejection murmur at the upper left sternal border with radiation to the back. Infants with critical pulmonary stenosis will be cyanotic and have ductal-dependent pulmonary blood flow. This critical form of stenosis may also be included in the category of cyanotic CHD with decreased pulmonary blood flow due to a large obligatory right-to-left atrial shunt. Newborns with critical pulmonary stenosis need to be treated with PGE_1 infusions. Balloon valvuloplasty of the pulmonic valve, performed in the cardiac catheterization lab, is usually successful in the immediate neonatal period. Surgery is considered only in those with severely dysplastic and/or hypoplastic valves or those with additional obstruction at the subvalvular or supravalvular level.

B. Cyanotic Congenital Heart Disease with Increased Pulmonary Blood Flow

Cardiac lesions with cyanosis and increased pulmonary blood flow can be classified further into those with increased pulmonary arterial vascularity in which there is hilar prominence on the chest radiograph (transposition of the great arteries and truncus arteriosus), and those with increased pulmonary venous vascularity in which a ground-glass or white-out appearance is seen on the chest radiograph (total anomalous pulmonary venous connection with pulmonary venous obstruction and hypoplastic left heart syndrome). In clinical practice, these two groups often cannot be accurately differentiated based on signs, symptoms, or radiographic findings, and echocardiography is needed to make the diagnosis.

1. Transposition of the Great Arteries

In dextro-transposition of the great arteries (d-TGA), the aorta arises from the RV while the pulmonary artery arises from the LV. The pulmonary and systemic circulations are configured in parallel rather than in series, resulting in cyanotic blood circulating back to the RV, which then ejects this blood into the systemic circulation while oxygenated blood returns to the LV, only to be ejected back to the pulmonary circulation. Cyanosis usually appears while the neonate is still in the delivery room, and cardiac examination may otherwise be normal. The chest radiograph may be normal, although the cardiac silhouette may occasionally resemble an “egg on a string” as a result of the right ventricular predominance and a narrow superior mediastinum related to the anterior-posterior

relationship to the great vessels. Pulmonary markings are normal or slightly increased. In newborns with d-TGA, initiation of PGE_1 infusion (0.05 µg/kg/min) can help increase oxygen saturation while awaiting balloon septostomy as a more definitive palliation. Balloon atrial septostomy (BAS) is often necessary to alleviate profound cyanosis, because mixing occurs most optimally between the low-pressure atria rather than at the ductus level. Furthermore, BAS is effective in decompressing the left atrium, which is often distended as pulmonary blood flow increases in the first few days after birth. However, neonates who undergo BAS are at increased risk of adverse neurologic outcomes, and some centers are performing the procedure only in deeply cyanotic patients.

Many neonates undergo surgical repair of the defect in the first week after birth, although palliation with BAS allows for stabilization and some flexibility in timing. Infants with d-TGA and a moderate to large VSD may not be diagnosed this early because interventricular mixing leads minimal cyanosis. Diagnosis may be delayed for weeks after birth, until findings of a large left-to-right shunt with signs of congestive heart failure (eg, poor feeding, tachypnea) develop. The arterial switch operation, in which the pulmonary artery is re-anastomosed to the RV and the aorta to the LV, with transfer of the coronary arteries, is now the surgical procedure of choice for the correction of d-TGA.

!

Transposition of the great arteries is the most common form of cyanotic heart disease to present on the first day after birth. It should be suspected in any cyanotic neonate, especially if poor response to supplemental oxygen is seen.

!

2. Truncus Arteriosus

Truncus arteriosus is a rare form of CHD in which a large single (truncal) valve provides a common outlet for the RV and LV. A large VSD allows for mixing of blood from both ventricles. The truncus arteriosus gives rise to the aorta, coronary arteries, and pulmonary arteries, and its classification depends on the location of the pulmonary artery or its branches as they arise. The mixing of saturated and unsaturated blood at the ventricular level and the ejection through the single truncal valve leads to mild cyanosis. Increased pulmonary blood flow with onset of congestive heart failure quickly develops as pulmonary vascular resistance drops. The chest radiograph shows a normal or increased heart size, with increased pulmonary markings and, in many cases, a right aortic arch. A significant percentage of these patients have a 22q11 microdeletion resulting in DiGeorge syndrome. Therefore, patients with truncus should be evaluated immediately for hypocalcemia (secondary to hypoparathyroidism) and T-cell immunodeficiency. Irradiated blood cells should be used for transfusions to prevent the development of graft-versus-host disease in patients in whom DiGeorge syndrome is suspected. Definitive surgical repair can usually be performed in the first week after birth. A valved homograft conduit is placed between the RV and the main pulmonary artery, after separating the branch pulmonary arteries from the truncus arteriosus, and the VSD is closed in such a way as to direct the left ventricular blood through the truncal valve into the aorta. Subsequent surgical procedures are necessary to increase the size of the conduit as the child grows.

Blood from both ventricles is ejected into the truncal artery. Systemic and pulmonary circulations mix, and the arterial oxygen saturation depends on the amount of pulmonary blood flow. As pulmonary vascular resistance decreases, pulmonary blood flow will increase, leading to LV overload, pulmonary edema, and decreased systemic oxygen delivery.

3. Total Anomalous Pulmonary Venous Connection

Patients with total anomalous pulmonary venous connection, a rare form of CHD, have their entire supply of oxygenated pulmonary venous blood returning to the systemic venous circulation. This admixture of oxygenated and desaturated systemic venous blood produces cyanosis present to the same degree in all four chambers of the heart. The four subtypes of total anomalous pulmonary venous connection are based on the location of pulmonary venous return: supracardiac (to the innominate vein or superior vena cava), intracardiac (to the coronary sinus or right atrium), infracardiac (below the diaphragm to the hepatic, portal, or umbilical venous system), and mixed (a combination of subtypes). The degree of obstruction to the pulmonary venous blood return determines the timing of presentation and the severity of cyanosis. Neonates with total anomalous pulmonary venous connection and significant obstruction frequently present in extremis with profound cyanosis. Those without obstruction are only mildly cyanotic and have a pulmonary flow murmur, so the diagnosis may be missed initially. Surgical repair is done immediately upon presentation with good long-term results.

Neonates with obstructed total anomalous pulmonary venous connection present with severe cyanosis.

Table 16-7 lists the causes of cyanosis in newborns, and **Table 16-8** outlines the initial evaluation and treatment of cyanotic newborns.

Table 16-7 CAUSES OF CYANOSIS IN NEWBORNS

Cyanotic heart disease

With decreased pulmonary vascularity

- Tetralogy of Fallot

- Tricuspid atresia

Critical pulmonary stenosis

With increased pulmonary blood flow

- Transposition of the great arteries
- Truncus arteriosus
- Total anomalous pulmonary venous connection

Respiratory diseases

Hematologic disorders (methemoglobinemia)

Neuromuscular disorders (hypoventilation)

Table 16-8 INITIAL EVALUATION AND TREATMENT OF CYANOTIC NEWBORNS

Initial Evaluation

Pulse oximetry screening (algorithm endorsed by American Academy of Pediatrics):

- Pass: oxygen saturation ≥95% in right hand and foot, and difference is ≤3 percentage points between right hand and foot.
- Fail: oxygen saturation <90% in right hand and foot.
- If oxygen saturation >90% and <95% in right hand and foot, or difference between right hand and foot is >3 percentage points, repeat screen in 1 hour. Rarely, 3 screens are needed (eg, the initial and 2 repeat screens, all separated by 1 hour)
- Fail: oxygen saturation is >90% and <95% in either right hand and foot, or if difference between right hand and foot >3 percentage points after third screen.

Chest radiograph

Electrocardiogram

Hyperoxia test (rarely performed due to the noninvasive pulse oximetry screen)

- Obtain Pao_2 on room air and after 10 minutes on 100% F_{IO_2}
- If preductal Pao_2 >150 mm Hg (>19.9 kPa), then no cyanotic congenital heart disease

Echocardiography: consult cardiology as appropriate for definitive diagnosis of suspected congenital heart disease.

Initial Treatment

Airway, breathing, circulation

Prostaglandin E_1 infusion at 0.05 µg/kg/min

Once diagnosis of cyanotic congenital heart disease is confirmed, titrate F_{IO_2} to achieve oxygen saturations appropriate for the specific lesion.

F_{IO_2}, fraction of inspired oxygen

IV. ACYANOTIC CONGENITAL HEART DISEASE

Case Study

A 3-month-old male infant is brought to the emergency department after several weeks of progressive tachypnea, diaphoresis, poor feeding, and decreasing activity. His vital signs include a pulse of 180 beats/min and respiratory rate of 75 breaths/min. A harsh systolic regurgitant murmur is detected. His liver is palpable 6 cm below the right costal margin.

Detection

– What are the most important initial steps in evaluating this infant?

– What should be included in the differential diagnosis?

– What is the most likely diagnosis?

Intervention

– Which studies and treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– Who needs to be aware of this patient?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, when, and in what order?

A. Acyanotic Congenital Heart Disease with Increased Pulmonary Blood Flow

1. Ventricular Septal Defect

VSD is the most common form of CHD. Its signs and symptoms are the result of the amount of left-to-right ventricular shunting. The size and direction of the shunt depend on the relative pressures and/or resistances of both the systemic and pulmonary circulations. Neonates with VSD are often asymptomatic until the pulmonary vascular resistance drops at 2 to 6 weeks of age, resulting in an increased shunt and the appearance of clinical symptoms.

An infant with a small to moderate VSD will present with a holosystolic murmur and remain asymptomatic. The intensity of the murmur is inversely proportional to the size of the defect. Children with larger VSDs have less impressive murmurs and typically develop signs and symptoms of heart failure (**Tables 16-2** and **16-3**). With these large defects, RV and pulmonary artery pressure will increase to systemic levels. Smaller defects create a pressure difference between the LV and RV.

With advances in the medical and surgical care of children with CHD, cardiothoracic surgery can now be performed safely, even in small infants. Percutaneous transcatheter closure is used in muscular and residual postoperative VSDs. Although the technique is safe and effective, patient size is a limitation. Indications for surgical closure include the following:

- Uncontrolled congestive heart failure
- Increased pulmonary vascular resistance with a risk for pulmonary vascular obstructive disease
- Failure to thrive despite maximum medical therapy
- Recurrent pulmonary infections
- Endocarditis
- Paradoxical emboli

Eisenmenger syndrome develops if a significant left-to-right shunt lesion remains unrepaired and long-standing pulmonary hypertension progresses to irreversible pulmonary vascular obstructive disease with resultant right-to-left ventricular shunting and cyanosis. Death follows as a result of pulmonary hemorrhage, infection, and/or paradoxical emboli.

2. Atrial Septal Defect

Atrial septal defect (ASD) is present in 10% of children with CHD. It is found more frequently in females than males (at a ratio of 2:1). ASDs occur in 3 separate locations in the atrial septum:

- *Ostium primum* defects are located in the lower third of the atrial septum, near the atrioventricular valves.
- *Ostium secundum* defects are found in the middle portion of the septum and are the most common.
- *Sinus venosus* defects occur in the posterior portion of the septum, adjacent to the venae cavae.

The direction and amount of atrial level shunting is determined by the relative compliances of the RV and LV and the size of the defect. Typically, the RV is more compliant, so left-to-right shunting occurs more frequently.

With rare exception, patients with an ASD are asymptomatic and classically have a fixed split S_2 with a systolic ejection murmur at the upper left sternal border due to relative pulmonary stenosis. Symptoms may develop later in childhood and are usually related to atrial arrhythmias. Indications for closure of an ASD are right ventricular volume overload, arrhythmias, paradoxical emboli, and

elevated pulmonary vascular resistance. Closure may be performed by placing an ASD occlusion device during cardiac catheterization or by suturing the defect primarily or with a patch. Both approaches are associated with low morbidity and mortality rates. Catheter-based closures are more successful in older children with ostium secundum defects. An adequate rim of atrial tissue around the defect is required to safely seat the device.

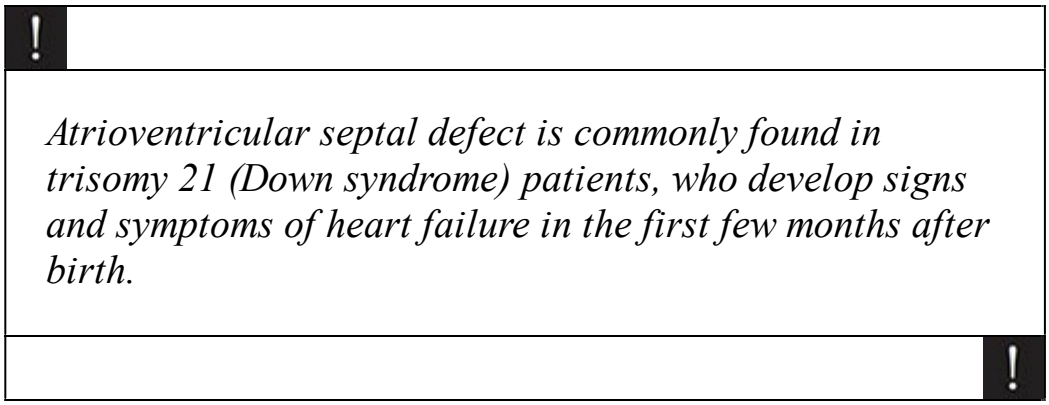
3. Patent Ductus Arteriosus

As an isolated anomaly, PDA occurs in approximately 1 in 2000 to 2500 live births. Its incidence increases in premature infants and is inversely proportional to the gestational age. The ductus arteriosus, an embryologic structure connecting the main pulmonary artery to the descending aorta, normally closes within the first few days after birth. Patients with PDA have left-to-right shunting dependent on the same issues of anatomic shunt size and relative resistance that govern VSD flow. In the presence of increased pulmonary vascular resistance, shunting through the PDA may be bidirectional. Infants with a large PDA, especially premature infants, have symptoms of heart failure. Those with a small to moderate PDA will present with an asymptomatic continuous murmur over the upper left sternal border.

Indomethacin and ibuprofen can be used to successfully close many PDAs in premature infants, although of the two, IV ibuprofen has fewer significant side effects. In older patients, catheter-based closure or surgical ligation is extremely effective, with essentially no morbidity or mortality. In the catheterization lab, a PDA occlusion device or multiple coils are placed to close the PDA.

4. Atrioventricular Septal Defect

Atrioventricular septal defect occurs in 7% of children with CHD. It is commonly found in children with trisomy 21 defect (Down syndrome). The lesion consists of a large defect in the atrioventricular septum with a common atrioventricular valve that is the inlet to both ventricles. Children with this defect are usually diagnosed in the first weeks after birth due to a systolic regurgitant murmur. If undiagnosed, patients will usually develop typical signs and symptoms of heart failure in their first months, as pulmonary vascular resistance decreases, and the degree of left-to-right shunting increases. To avoid acute and long-term consequences of pulmonary hypertension, surgical repair is required in infancy, ideally at 3 to 6 months of age.


!
Atrioventricular septal defect is commonly found in trisomy 21 (Down syndrome) patients, who develop signs and symptoms of heart failure in the first few months after birth.
!

The causes of acyanotic heart disease are listed in **Table 16-9**. An overview of the evaluation and treatment of acyanotic heart disease can be found in **Table 16-10**.

Table 16-9	CAUSES OF ACYANOTIC CONGENITAL HEART DISEASE

Acyanotic congenital heart disease with increased pulmonary blood flow (shunt lesions)
- Ventricular septal defect
- Atrial septal defect
- Patent ductus arteriosus
- Atrioventricular septal defect

Acyanotic congenital heart disease with obstruction
- Pulmonary stenosis (valvular, supravalvular, subvalvular) without an atrial or ventricular septal defect
- Aortic stenosis (valvular, supravalvular, subvalvular)
- Coarctation of the aorta

Other causes of heart failure
- Arrhythmia
- Cardiomyopathy
- Myocarditis
- Coronary artery anomalies
- Isolated atrioventricular valve insufficiency (mitral regurgitation, which can be associated with Marfan disease, tricuspid regurgitation)

Table 16-10	INITIAL EVALUATION AND TREATMENT OF ACYANOTIC HEART DISEASE

Initial Evaluation

Chest radiograph

Electrocardiogram with rhythm strip

Echocardiography

Initial Treatment

Diuretics
- Loop diuretic (furosemide, 1-2 mg/kg/dose orally or IV every 6-12 h)
- Thiazides (chlorothiazide, 2-5 mg/kg/dose IV every 12 h)
- Potassium-sparing agent (spironolactone, 0.5-3 mg/kg/day divided, every 6-24 h)

Afterload-reducing agents (should not be used in patients with unrepaired LVOTO (ie, aortic stenosis)
- ACE inhibitors: enalaprilat, 5-10 µg/kg/dose IV every 8-12 h; enalapril, 0.05-0.25 mg/kg/day divided, every 12-24 h orally)
- Milrinone, 0.25-0.75 µg/kg/min IV
- Nitroprusside, 0.5-10 µg/kg/min IV
- Nitroglycerine, 0.5- 5 µg/kg/min IV

Inotropic agents
- Digoxin, 8-10 µg/kg/day divided every 12 h (dose is age dependent)
- Dopamine, 5-10 µg/kg/min (adrenergic agonist)
- Dobutamine, 5-15 µg/kg/min (adrenergic agonist)
- Epinephrine/adrenaline, 0.05-0.1 µg/kg/min (adrenergic agonist)

- Milrinone, 0.25-0.75 μg/kg/min (phosphodiesterase inhibitor)

NOTE: Optimal use of oxygen; hyperoxia can increase left-to-right shunting.

IV, intravenous; ACE, angiotensin-converting enzyme; LVOTO, left ventricular outflow tract obstruction

V. LOW CARDIAC OUTPUT SYNDROME

Case Study

A 4-year-old boy is seen in the emergency department with lethargy 2 weeks after an uncomplicated surgical repair of an atrial septal defect. He is found to be tachycardic (heart rate, 130 beats/min), tachypneic (respiratory rate, 40 breaths/min), and hypotensive (blood pressure, 75/60 mm Hg) with muffled heart sounds on auscultation. An arterial blood gas measurement shows a marked lactic acidosis.

Detection

– What are the most important initial steps in evaluating this child?

– What should be included in the differential diagnosis?

– What is the most likely diagnosis?

Intervention

– What studies and treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need other therapeutic interventions?

Effective Communication

– Who needs to be aware of this patient?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, when, and in what order?

Low cardiac output syndrome (LCOS) describes a clinical and biochemical state of inadequate systemic oxygen delivery to meet the patient's metabolic demands. The syndrome is frequently seen in patients with severe sepsis, myocarditis, and cardiomyopathies and after pediatric cardiac surgery. Postoperative physiologic changes in response to cardiopulmonary bypass, residual lesions, cardioplegia, ventriculotomy, changes in the loading conditions of the myocardium, or myocardial ischemia during aortic cross-clamping all may contribute to the development of LCOS.

When unrecognized or inadequately treated, LCOS can lead to irreversible end-organ failure, cardiac arrest, and death. Although precise and accurate assessment of oxygen delivery in critically ill children is challenging, hemodynamic and biochemical parameters can guide the clinician at the bedside. Arterial lactate and central venous oxygen saturation are two important biochemical markers in patients with LCOS.

Common causes of low cardiac output are presented in **Tables 16-11** and **16-12**. The findings associated with LCOS are listed in **Table 16-13**, but a patient need not have all findings to meet the diagnostic criteria. The goal is to make the diagnosis early so as to identify treatable causes and institute timely therapy to prevent irreversible end-organ injury and death.

Table 16-11 CAUSES OF POSTOPERATIVE LOW CARDIAC OUTPUT

- Arrhythmias
- Hypovolemia
- Anemia
- Myocardial dysfunction
- Residual cardiac lesions
- Pulmonary hypertension
- Cardiac tamponade

Table 16-12 POSSIBLE RESIDUAL LESIONS AFTER SURGERY

- Valvular stenosis
- Valvular insufficiency
- Arterial stenosis
- Residual shunts
- Outflow tract obstruction

Table 16-13 SIGNS AND SYMPTOMS OF LOW CARDIAC OUTPUT SYNDROME

- Tachycardia
- Poor peripheral effusion
- Decreased urine output
- Lactic acidosis
- Elevated or rapidly rising lactate levels (>0.75 mmol/L/h [>6.76 mg/dL])
- Widened arterial to mixed venous oxygen saturation difference (>30%-40%)
- Altered mental status
- Late findings
  - Increased creatinine
  - Increased liver enzymes
  - Seizures
  - Hypotension

A. Arrhythmias

The development of LCOS after surgery should prompt a reevaluation for the presence of residual hemodynamic lesions, tamponade, or arrhythmia.

An unstable patient with acute onset of a wide complex tachycardia should be presumed to have ventricular tachycardia and be treated immediately with direct-current biphasic defibrillation at 2-4 J/kg.

The cardiac rhythm must be assessed in an unstable postoperative patient. A 12-lead electrocardiogram with a long rhythm strip should be performed as soon as possible. The most common arrhythmias following congenital heart surgery and their usual modes of treatment are presented in **Table 16-14**. Electrocardiographic images are shown in **Figures 16-2** through **16-5**. These rhythms can also occur without any underlying structural heart disease, as in myocarditis or Kawasaki disease.

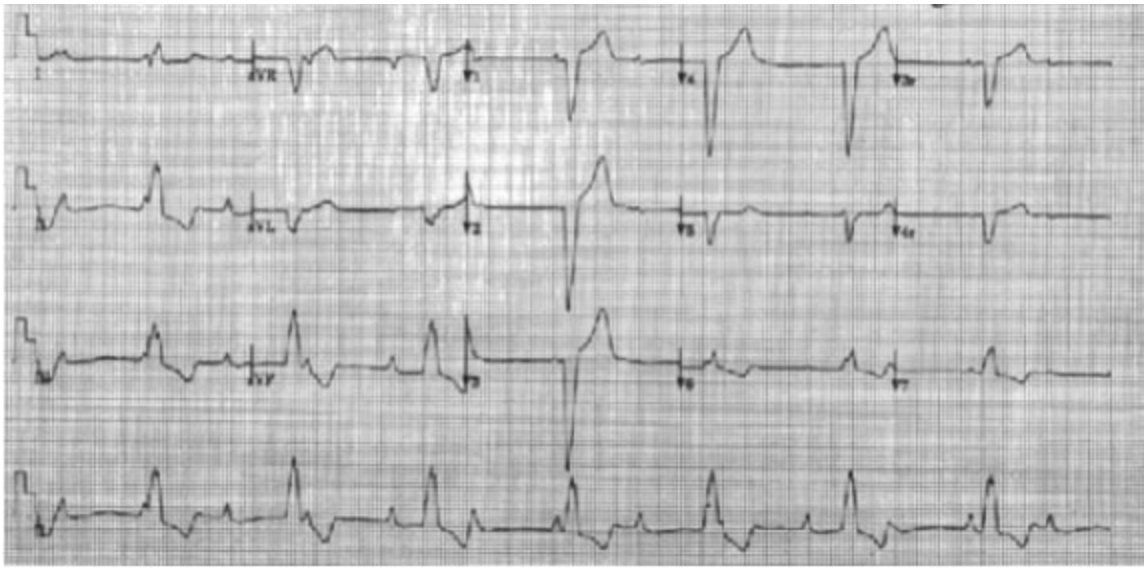
Table 16-14 COMMON ARRHYTHMIAS FOLLOWING CONGENITAL HEART SURGERY AND USUAL TREATMENTS

Arrhythmia	Signs/Symptoms	Circumstances	Treatment
Junctional ectopic tachycardia	Narrow (normal) complex tachycardia Rate ≥160 beats/min AV disassociation is most common	Most common after surgeries involving the ventricular septum: Tetralogy of Fallot VSD Atrioventricular septal defect	Pace the atrium to restore AV synchrony Treat hyperthermia, agitation Minimize β-adrenergic dosing Amiodarone (5 mg/kg/dose IV over 20-60 min; may need to repeat) Procainamide (3-6 mg/kg/dose IV)
Heart block	*First degree:* prolonged PR interval *Second degree:* Type I/Wenckebach: progressive prolongation of the PR interval until AV conduction is lost Type II: AV conduction that occurs in a fixed ratio other than 1:1 *Third degree:* complete loss of AV conduction resulting in bradycardia with AV disassociation	Most common after surgeries around AV node: VSD repairs Mitral valve replacement Enlargement of left ventricular outflow tract (Konno procedure) L-looped heart (ventricular inversion) is additional risk factor	None (first degree and Wenckebach often asymptomatic) Temporary or permanent pacing Second degree, type II, and third degree block: always require dual chamber pacing If dual chamber pacing is not possible, ventricular pacing (transthoracic postoperative temporary epicardial wires, transcutaneous pacing pads, transvenous via central vein into heart) will prevent ventricular asystole.
Orthodromic reciprocating	Reentrant tachycardia (normal conduction from	Wolff-Parkinson White syndrome Concealed accessory pathway	Adenosine (100-300 µg/kg IV rapid push) Synchronized direct current cardioversion

tachycardia	atria to ventricles across AV node with atrial reentry via an accessory pathway) with P in T wave VA relationship always 1:1 Sudden onset and cessation of tachycardia		(0.5-2 J/kg) Atrial overdrive pacing Amiodarone or ß-blocker (atenolol) for recurrent episodes
Atrial flutter/ fibrillation	Reentrant tachycardia within the atrium Narrow QRS complex, usually with 2:1 or 3:1 AV conduction	Most common with severe right atrial enlargement or extensive atrial surgery: Fontan procedure (atriopulmonary connection or lateral tunnel) years after surgery Ebstein anomaly Mustard procedure (for transposition of great arteries) Senning procedure (for transposition of great arteries)	Adenosine (does not alter atrial rate, but can help in diagnosis) Synchronized cardioversion (0.5-2 J/kg) Rapid atrial pacing (at rate greater than atrial rate) Amiodarone or other antiarrhythmics (for recurrent episodes)
Ventricular tachycardia	Wide complex tachycardia Rare in pediatrics	Most common in: Failing ventricles with poor systolic function Severely hypertrophic cardiomyopathy	Direct current cardioversion (2-4 J/kg) Amiodarone (5 mg/kg IV) Lidocaine (0.5-1.5 mg/kg IV)

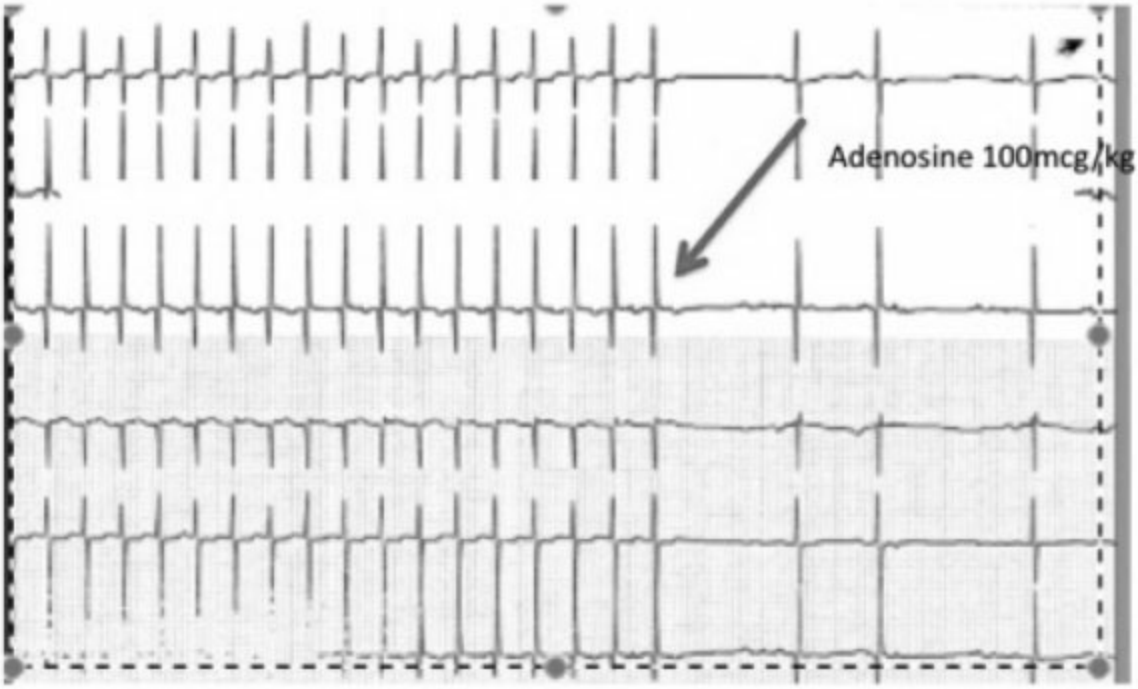
VA, ventricular to atrial; AV, atrioventricular; VSD, ventricular septal defect

Figure 16-2. Third Degree Atrioventricular Block



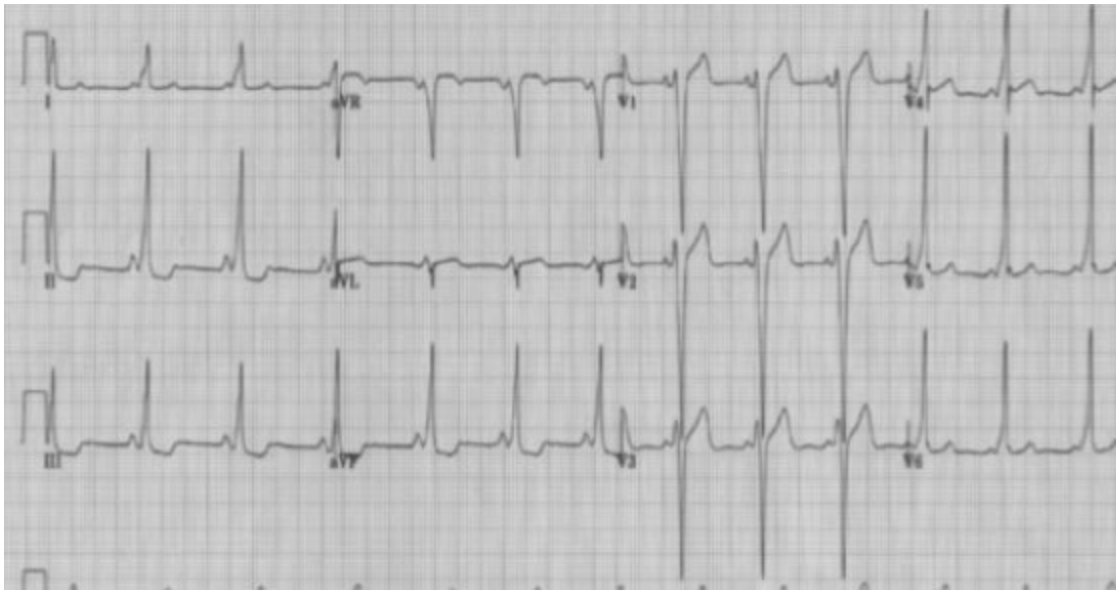
Complete atrioventricular block (third degree), either congenital or acquired (postoperative). Lack of conduction between the atria and the ventricles is seen as a slow QRS rate and a regular atrial rhythm with an irregular PR interval. Courtesy of Ana Lia Graciano, MD, FAAP, FCCM.

Figure 16-3. Supraventricular Tachycardia



In paroxysmal supraventricular tachycardia (SVT), the electrocardiogram demonstrates regular RR interval and narrow QRS. SVT is usually caused by a re-entry circuit involving the atrioventricular node; adenosine causes a temporary block in the node, interrupting the re-entry circuit.
Courtesy of Ana Lia Graciano, MD, FAAP, FCCM.

Figure 16-4. Wolff-Parkinson-White Syndrome



In Wolff-Parkinson-White syndrome with the patient in tachycardia (heart rate >200 beats/min), the impulse is carried in an antegrade manner (known as *orthodromic conduction*) through the atrioventricular node, resulting in a normal QRS complex, and in retrograde manner via the accessory pathway to the atrium, perpetuating the narrow complex tachycardia. Alternatively, in antidromic conduction in the tachycardia, the impulse from the atrium is conducted through the accessory pathway directly into the ventricle, resulting into a wide complex tachycardia which can be fatal. The circuit is completed with retrograde conduction through the atrioventricular node back into the atrial. The typical electrocardiogram pattern with an upstroke of the QRS (delta wave) is recognized after cessation of the tachycardia.
Courtesy of Ana Lia Graciano, MD, FAAP, FCCM.

Figure 16-5. Atrial Flutter

Atrial flutter is characterized by rapid, regular sawtooth flutter waves at rates between 250 to 500 per minute. Atrioventricular conduction is blocked to some degree, resulting in the ventricle responding irregularly every 2 to 4 atrial beats.
Courtesy of Ana Lia Graciano, MD, FAAP, FCCM.

B. Cardiac Tamponade

One important cause of LCOS that requires immediate recognition and management is cardiac tamponade, a clinical syndrome caused by the accumulation of fluid in the pericardial space and leading to impaired ventricular filling and subsequent hemodynamic compromise. Echocardiography can confirm the diagnosis by demonstrating a collection of pericardial fluid with associated atrial compression or ventricular compression beginning first on the right side of the heart and progressing to the left side with increasing severity. The diagnosis of tamponade must be considered in any postoperative patient with LCOS. Cardiac tamponade is treated by clearing occluded chest tubes, by pericardiocentesis, or by mediastinal re-exploration. Rapid fluid resuscitation may be necessary to support the circulation until adequate pericardial drainage is achieved.

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Cardiac tamponade must be considered in all patients with LCOS after open heart surgery.

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C. Myocardial Dysfunction

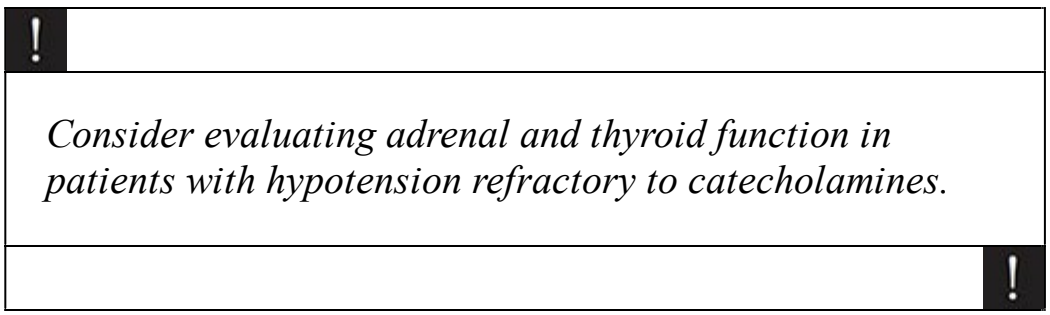
Myocardial contractility can be enhanced by using β-adrenergic agonists or phosphodiesterase inhibitors through different mechanisms with the final common pathway (**Table 16-10**) being rapid calcium influx. This leaves the sarcoplasmic reticulum to bind with troponin C, resulting in actin/myosin cross-bridging and contraction. β-adrenergic agonists work by increasing intracellular cyclic adenosine monophosphate, while phosphodiesterase inhibitors serve to increase cyclic guanosine monophosphate, both of which result in increased calcium release from the sarcoplasmic reticulum. Diastolic relaxation is an energy-requiring step with reuptake of calcium into the sarcoplasmic reticulum.

Milrinone, a selective phosphodiesterase III inhibitor, increases cyclic guanosine monophosphate

concentrations, leading to an increase in intracellular calcium and contractile force in cardiac muscle. At infusion rates of 0.25 to 0.75 μg/kg/min given in a dose-related manner, milrinone has been shown to prevent LCOS following surgical repair of CHD. Phosphodiesterase inhibition is not β-adrenergic receptor dependent; therefore, it is not altered by receptor downregulation that occurs during chronic adrenergic stimulation. Another advantage of phosphodiesterase inhibitors is that they promote afterload reduction for the systemic (and pulmonary) ventricle, hence the term *inodilators*. Finally, they improve diastolic relaxation (lusitropic properties), improving the diastolic dysfunction common in infants and children with CHD.

Blood pressure is determined by the interplay between cardiac output (which is the product of heart rate and stroke volume) and systemic vascular resistance (SVR). Stroke volume is dependent on preload, afterload (ie, SVR), and contractility. Therefore, use of vasodilators for afterload reduction results in an increase in cardiac output and oxygen delivery, albeit often at a lower blood pressure. When vascular tone is high, either intrinsically or secondary to the use of increased doses of adrenergic agonists, and cardiac output is low, pure vasodilating agents can be used to improve cardiac output and oxygen delivery. Nitroprusside (0.5-10 μg/kg/min) or nitroglycerine (0.5-5 μg/kg/min) are commonly used for this purpose. Likewise, synergistic improvements in cardiac output can be obtained with the addition of low-dose epinephrine (0.01-0.05 μg/kg/min), which increases contractility and heart rate without significant increases in afterload. Caution should be used with prolonged nitroprusside infusion, particularly with acute kidney injury, as cyanide toxicity can develop.

When blood pressure is inadequate to maintain sufficient perfusion, cardiac output should be increased before increasing SVR. Commonly used vasopressors (with α-adrenergic agonist effects) include epinephrine and norepinephrine, with the latter being most potent. Vasopressin (0.0003-0.002 U/kg/min), which binds to the V receptor without adrenergic receptor activation, is a useful adjunct in increasing SVR. Finally, phenylephrine, a pure α-agonist, can be used when necessary. Persistent hypotension refractory to fluids and catecholamines may indicate adrenal insufficiency, which should be quickly diagnosed with levels before and after corticotropin stimulation and treated with stress-dose hydrocortisone.



The neonatal heart has an immature calcium-handling system and is dependent on extracellular calcium for contractility. Clinical evidence of improved hemodynamics following neonatal cardiac surgery can be attained by maintaining ionized calcium levels at the 90th percentile of normal. Hypocalcemia, either secondary to genetic issues (such as chromosome 22 deletions) or as a result of citrate in packed red blood cells, should be aggressively treated, usually with CaCl (10-20 mg/kg IV) or with continuous calcium infusions.

Optimal management of the pediatric patient with cardiac disease requires a comprehensive

understanding of the basic principles of oxygen delivery, cardiovascular physiology, and the anatomy and physiology of CHD. Signs and symptoms of LCOS should be treated aggressively, and diagnostic and therapeutic strategies should address both common and lesion-specific problems.

The tests used to evaluate patients with low cardiac output are outlined in **Table 16-15**.

Table 16-15	EVALUATION OF PATIENTS WITH LOW CARDIAC OUTPUT
Central venous and arterial pressure monitoring	
Arterial and venous blood gas analysis	
Hemoglobin levels	
Serum electrolytes (including Ca^{++}, Mg^{++}, $PO4^{-}$)	
Serum lactate	
12-lead electrocardiogram with rhythm strip	
Chest radiograph	
Echocardiogram	

Key Points

Management of the Child With Congenital Heart Disease

- Infants develop low cardiac output or shock in the first weeks of life must be evaluated quickly for congenital heart disease.
- Congenital heart disease can be categorized into 2 groups—acyanotic and cyanotic—and a presumptive diagnosis can be made by integrating information obtained from the history, physical examination, chest radiograph, and pre- and post-ductal pulse oximetry. A definitive diagnosis can be made based on echocardiogram results, with a subset of patients requiring diagnostic cardiac catheterization.
- Initiation of a prostaglandin E_1 infusion can be lifesaving in neonates with congenital heart disease and ductal-dependent blood flow, so this infusion should be considered in any neonate who with shock or cyanosis.
- Prompt recognition and appropriate treatment of the low cardiac output syndrome is lifesaving and essential for preserving organ function in pediatric patients with congenital heart disease.

Suggested Readings

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ONCOLOGIC AND HEMATOLOGIC EMERGENCIES AND COMPLICATIONS

Objectives

- Detail risk factors unique to the immunosuppressed pediatric patient.
- Recognize and treat causes of shock in the pediatric cancer patient.
- Recognize and treat causes of respiratory failure in the pediatric cancer patient.
- Review metabolic abnormalities associated with tumor lysis syndrome and outline the treatment priorities.
- Describe risk factors associated with hyperleukocytosis.
- Identify and treat spinal cord compression.
- Recognize and treat causes of disseminated intravascular coagulation.
- Describe blood product use and its complications

Case Study

A 12-year-old girl with acute lymphocytic leukemia, who was recently released from the hospital after induction chemotherapy, presents to a referring hospital with a 2-hour history of fever, shivering, and chills after having her central line flushed with heparin. On admission, her temperature is 39.2°C (102.6°F), heart rate is 156 beats/min, blood pressure is 82/35 mm Hg, and respiratory rate is 28 breaths/min. Laboratory data obtained on admission show a white blood cell (WBC) count of 0.1 x 10^9 cells/L, hemoglobin 7.2 g/dL (72 g/L), and platelet count of 45,000 x 10^9 cells/L. Her electrolytes are normal except for a K^+ of 2.8 mEq/L (2.8 mmol/L). Blood culture results 12 hours after identify Gram-positive cocci. You have been asked to evaluate the patient.

Detection

– What is this patient's physiologic status?

– What is the most likely diagnosis?

Intervention

– Which treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does the patient need additional fluids? Is a blood transfusion indicated?

Effective Communication

– When the patient's clinical status changes, who needs to know this information and how will it be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, where, and when?

I. INTRODUCTION

This chapter is designed to provide essential information on aspects of care that may be critical in the management of medical complications in the pediatric oncology/hematology patient. Recent evidence supports the use of aggressive therapies early in the course of the patient's acute illness. These may include broad-spectrum antimicrobial coverage, fluid resuscitation, and early respiratory, vasopressor, and/or inotropic support. The therapeutic goals should be discussed with the primary attending clinician, the patient's guardian, the patient (if appropriate), and the ICU team. Decisions concerning continuation or limitations of care should be frequently explored based on the patient's primary diagnosis, oncologic prognosis, and clinical course.

II. FEVER AND NEUTROPENIA

A. Definitions

Neutropenic fever, a common complication seen during the treatment of pediatric malignancies, is defined as a single oral temperature >38.3°C (>100.9°F) or a sustained temperature over >38°C (>100.4°F) for ≥1 hour in a patient with an absolute neutrophil count of either <500/mm³ or <1000/mm³ with a downward trend on 2 separate peripheral blood counts. Rectal temperature measurements are contraindicated in neutropenic patients due to the potential disruption of the rectal mucosal barrier, which creates a route for infection.

B. Risk Factors

The International Pediatric Fever and Neutropenia Guideline panel recommends risk stratification to identify patients at high risk of complications based on various patient, disease, and episode specific factors (**Table 17-1**). Patients without any high risk factors are considered to be at low risk. Various validated scales are available, but no single tool works across various settings. Each institution should develop a scheme based on available resources.

Table 17-1	FACTORS ASSOCIATED WITH HIGH RISK OF COMPLICATIONS IN PATIENTS WITH FEBRILE NEUTROPENIA
Specific to patient and disease	
Infants Acute myeloid leukemia Burkitt lymphoma Progressive disease or relapse with marrow involvement Allogeneic hematopoietic stem cell transplant Near myeloablative chemotherapy Presence of central venous catheter	
Specific to episode	
Clinical indicators High fever ≥39°C (≥102.2°F) Hemodynamic instability Respiratory symptoms: tachypnea and hypoxia New onset abdominal pain Altered mental status Focal infections (eg, pneumonia, neutropenic enterocolitis, severe oral mucositis, perianal lesions)	
Laboratory indicators Prolonged neutropenia >7 days Absolute neutrophil count <100/mm³ Elevated C-reactive protein >10 mg/L Absolute monocyte count <100 /mm³ Platelet count <20,000 to 50,000 /mm³ Gram-negative bacteremia Liver or renal failure	

C. Treatment

The standard of care for patients with fever and neutropenia is immediate empiric inpatient IV antibiotic therapy within an hour of evaluation. Many institutions have created protocols to reduce time to antibiotic administration, but venous access, time waiting for laboratory results, and availability of the antibiotic remain some of the most common issues delaying this practice. The patient requires a thorough history and physical examination. Before initiating IV antibiotics, blood from all lumens of a central venous catheter should be cultured, and collection of a specimen for concurrent peripheral blood culture should be considered. Urinalysis and urine culture should be obtained if a midstream clean catch sample is easily available in patients who have not been catheterized. A culture specimen can be obtained from all indwelling catheters. Collection for a

blood culture should not unjustifiably delay antibiotic administration. Complete blood cell count, chemistry studies, C-reactive protein, procalcitonin, *Clostridium difficile* toxin, other stool studies, a chest radiograph, and spinal fluid culture should be obtained if clinically indicated. Early drainage of any fluid collection (abscess, effusion, and empyema) for culture and symptom relief is highly recommended.

The organisms associated with serious infections in children and the suggested antimicrobials and dosages are summarized in **Table 17-2**. High-risk febrile neutropenic patients without a focus of infection should be empirically treated with a single broad-spectrum antibiotic with antipseudomonal β-lactam spectrum or a fourth-generation cephalosporin (such as cefepime), or a very broad-spectrum antibiotic (such as meropenem). Dual Gram-negative coverage with aminoglycosides or a glycopeptide should be reserved for clinically unstable patients, for suspected resistant infections, or in centers with a high rate of resistant organisms. Dual Gram-negative coverage or empirical glycopeptide coverage should be discontinued in the absence of a specific microbiologic indication for treatment after 24 to 72 hours. Monotherapy should be continued. Empiric coverage may be discontinued after 48 hours of negative cultures, a 24-hour period without fever, and evidence of bone marrow recovery. High-risk patients with prolonged neutropenia who are receiving high-dose corticosteroids are at risk of invasive fungal infections. These patients should receive empirical antifungal therapy.

Table 17-2 TREATMENT RECOMMENDATIONS FOR SERIOUS INFECTIONS IN IMMUNOSUPPRESSED CHILDREN[a]

Organism	Treatment
Gram-positive organisms Coagulase-negative *Staphylococcus*, *Staphylococcus aureus*, α-hemolytic streptococcus, *Enterococcus, Corynebacterium*	Vancomycin[b]: 15 mg/kg every 6 h IV, maximum dose, 4 g/24 h Linezolid: 10 mg/kg every 8 h IV; maximum dose, 1.2 g/24 h Daptomycin[b]: 4-6 mg/kg every 24 h IV (not approved for children <18 years)
Gram-negative organisms *Klebsiella, Bacillus, Pseudomonas, Escherichia coli*	Cephalosporins[b]: – Cefepime: 50 mg/kg every 8 h IV/IM; maximum dose, 6 g/24 h – Ceftazidime[c]: 50 mg/kg every 8 h IV/IM; maximum dose, 6 g/24 h Aminoglycosides[b]: – Gentamicin: 2.5 mg/kg every 8 h IV – Tobramycin: 2.5 mg/kg every 8 h IV Carbapenems[b]: – Imipenem-cilastatin: 25 mg/kg every 6 h IV; maximum dose, 4 g/24 h – Meropenem: 40 mg/kg every 8 h IV; maximum dose, 6 g/24 h
Fungi *Candida, Aspergillus*	Fluconazole[b]: loading dose, 12 mg/kg IV; maintenance, 6 mg/kg IV every 24 h after loading dose; maximum loading dose, 400 mg Liposomal amphotericin B: 3-5 mg/kg every 24 h IV Caspofungin: loading dose, 70 mg/m² IV; maintenance, 50 mg/m² IV every 24 h after loading dose; maximum loading dose, 70 mg; maximum maintenance dose, 50 mg Voriconazole:

	− Children 2-12 years: 9 mg/kg IV every 12 h x 2 doses, then 8 mg/kg IV every 12 h (maximum dose, 350 mg every 12 h); monitor trough levels closely (>2 µg/mL) − >12 years and older: loading dose, 6 mg/kg every 12 h IV x 2 doses, then 4 mg/kg every 12 h IV; monitor trough levels closely (>1 µg/mL)
Anaerobes	Clindamycin: 10 mg/kg every 6-8 h IV/IM; maximum dose: 4.8 g/day Metronidazole: 7.5 mg/kg every 6 h IV/PO; maximum dose, 4 g/24 h
Antivirals – Respiratory syncytial virus – Influenza types A and B	Ribavirin: 6 g by aerosol (small-particle aerosol generator) over 12-18 h daily for 3-7 days (6 g in 300 mL preservative-free sterile water)[d] Zanamivir: patients older than 7 years – Day 1: 2 doses (5 mg via inhalation) at least 2-12 h apart – Days 2-5: 2 doses (5 mg via inhalation) every 12 h for 4 days; initiate within 2 days of symptom onset Oseltamivir[b]: – Infants ages 2 weeks to 12 months: 3 mg/kg PO twice daily for 5 days – Children ages 1-12 years: – <15 kg: 30 mg PO twice daily for 5 days – >15-23 kg: 45 mg PO twice daily for 5 days – >23-40 kg: 60 mg PO twice daily for 5 days – >40 kg: 75 mg PO twice daily for 5 days – Children older than 12 years: 75 mg PO twice daily for 5 days Peramivir[b]: − Patients ages 6-18 years: 10 mg/kg/dose IV once a day for 5 days (off-label use) − Patients older than 18 years: 600 mg IV once a day for 5 days
Antivirals – Parainfluenza – CMV pneumonia	Ribavirin may be beneficial if used early (see respiratory syncytial virus treatment for dose) Ganciclovir[b]: 5 mg/kg every 12 h IV OR Foscarnet[b]: 90 mg/kg every 12 h IV AND Immunoglobulin: 0.5 g/kg daily IV
Pneumocystis jiroveci	Trimethoprim-sulfamethoxazole[b]: 5 mg/kg trimethoprim component every 6-8 h IV Methylprednisolone: 1 mg/kg every 12 h; should be started within 72 h in patient presenting with hypoxia (Pao_2 <70 mm Hg [9.3 kPa] on room air)

IM, intramuscular; PO, orally; CMV, cytomegalovirus

[a]These recommendations are general guidelines only. Specific antibiotic choices should be individualized, taking into consideration clinical circumstances (renal function, liver function), patient age, immunization status, and local microbial virulence, sensitivities, and patterns of resistance. The antibiotic dosing, interval, and frequency should be discussed with a pediatric intensivist or pediatric infectious disease expert.

[b]Adjust for renal failure.

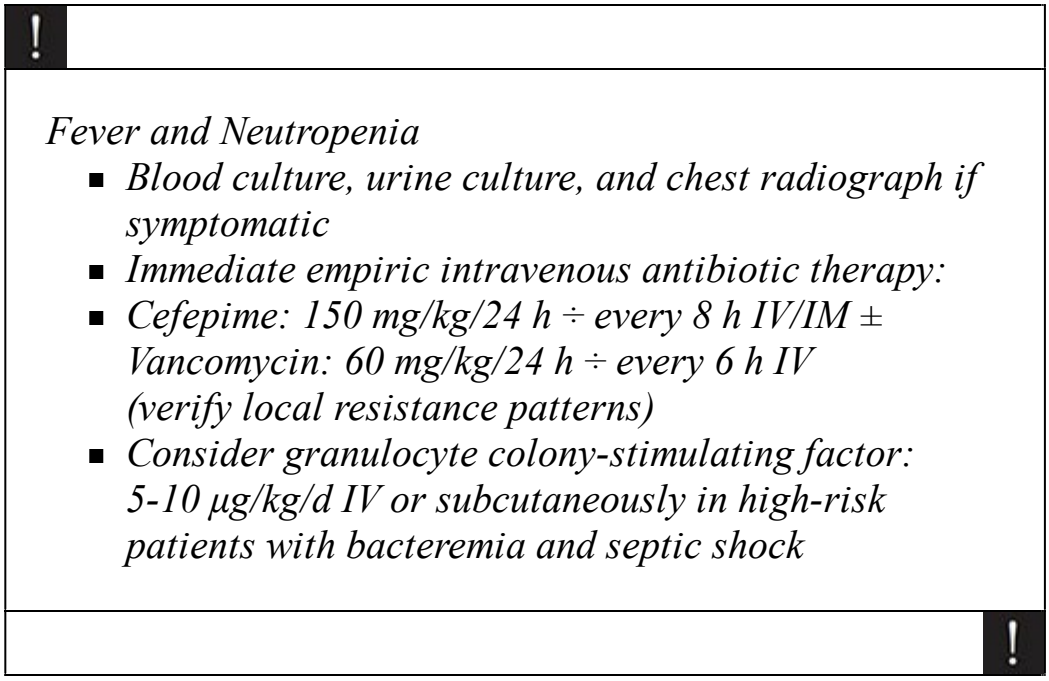
[c] Not recommended as an empiric monotherapy due to increasing resistance of Gram-negative bacteria.

[d]Reserved for severe cases.

The removal of the IV device is warranted following a diagnosis of a catheter-related infection. Alternate IV access should be considered for patients with hemodynamic instability, persistent bacteremia, infections caused by fungi, mycobacteria, *Staphylococcus aureus*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, *Stenotrophomonas maltophilia*, vancomycin-resistant

Enterococcus, and Gram-negative infections.

Colony-stimulating factors may be used in high-risk patients to shorten the duration of neutropenia. In addition, these agents may help reduce the length of antibiotic treatment and hospital stay. They have been utilized with increasing frequency for patients with neutropenia and multiple organ system failure, sepsis or septic shock, and/or invasive fungal disease. Granulocyte colony-stimulating factor given IV or subcutaneously in a dose of 5 to 10 μg/kg/d stimulates the release of neutrophils. Granulocyte macrophage colony-stimulating factor in a dose of 5 μg/kg/d IV or subcutaneously enhances the release of neutrophils and macrophages and may be considered for patients in whom fungal disease is confirmed or strongly suspected. Either medication should be continued until the absolute neutrophil count exceeds 1000/mm³ for 3 consecutive days.


!
Fever and Neutropenia
Blood culture, urine culture, and chest radiograph if symptomatic
Immediate empiric intravenous antibiotic therapy:
Cefepime: 150 mg/kg/24 h ÷ every 8 h IV/IM ±
Vancomycin: 60 mg/kg/24 h ÷ every 6 h IV
(verify local resistance patterns)
Consider granulocyte colony-stimulating factor:
5-10 μg/kg/d IV or subcutaneously in high-risk patients with bacteremia and septic shock
!

D. Complications of the Febrile Neutropenic State

1. Bacteremia

Documented bacteremia in febrile neutropenic patients occurs at a rate of 10% to 45%, with catheter-related infections being the most frequent cause. In the United States, the most common infective agents are coagulase-negative staphylococci, *S aureus*, and enterococci. Patients with acute myelogenous leukemia have a high risk for *Streptococcus viridans* infections. The rate of infections caused by Gram-negative organisms appears to be increasing worldwide. In a retrospective study in Asia, 53.9% of all bacteremias in children with fever and neutropenia were caused by Gram-negative organisms, with the most common being *Escherichia coli* and *Klebsiella pneumoniae*.

2. Lower Respiratory Tract Infections

Case Study

An 11-year-old boy who underwent peripheral blood stem cell transplant 28 days ago is admitted to the ICU from the bone marrow transplant ward because of respiratory distress and hypoxia. His oxygen saturation was 89% on a fraction of inspired oxygen of 0.5. A chest computed tomographic (CT) scan shows a diffuse ground-glass pattern. His WBC count is 200/mm³.

Detection

– What is this child's physiologic status?

– What is the most likely diagnosis?

Intervention

– Which treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Is the patient a candidate for noninvasive mechanical ventilation? Would you intubate now?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will the information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, where, and when?

The immune deficiency caused by the primary disease and/or the toxicity of the treatment make pediatric cancer patients susceptible to a wide range of infectious and noninfectious problems that may progress rapidly to respiratory failure. Initiation of broad-spectrum antibacterial, antifungal, and antiviral therapy is often indicated in patients with rapid clinical or radiologic deterioration. High-resolution CT should be considered in persistently febrile patients (>96 hours) and in all patients with respiratory symptomatology, irrespective of the plain radiograph findings. Bronchoscopy with bronchoalveolar lavage or open lung biopsy should be considered early in the course of the patient's illness to differentiate infectious from noninfectious etiologies (diffuse alveolar hemorrhage), particularly in patients who have undergone bone marrow and stem cell transplantation.

Community-acquired viral infections, such as influenza, respiratory syncytial virus, and parainfluenza, can have devastating effects in pediatric oncology patients, and transplant patients in particular. Patients with respiratory syncytial virus infections may benefit from ribavirin, palivizumab, and immunoglobulin. Those with influenza infections will benefit from the early use of

antiviral medications (**Table 17-2**). No effective therapy exists for parainfluenza pneumonia. The mortality rate for cytomegalovirus (CMV) pneumonitis in bone marrow transplant patients is high. The recommended initial treatment for CMV pneumonia is a combination of IV ganciclovir and immunoglobulin. *Pneumocystis jiroveci* (formerly *Pneumocystis carinii*) and CMV pneumonia should be included in the differential diagnosis of any immunocompromised patient who presents with respiratory symptoms, hypoxemia, and diffuse patchy infiltrates on the chest radiograph. The treatment of choice for *P jiroveci* pneumonia is IV trimethoprim-sulfamethoxazole (pentamidine in allergic patients). Corticosteroids (methylprednisolone, 1-2 mg/kg/d IV given every 6 hours) should be initiated in patients with moderate or severe *P jiroveci* pneumonia (Pao_2 <70 mm Hg [9.3 kPa] on room air) within 72 hours of initiating antibiotic therapy.

Pediatric oncology patients presenting with respiratory failure should promptly receive noninvasive positive pressure ventilation or be intubated and mechanically ventilated using lung protective strategy as indicated. Early institution of high-frequency oscillatory ventilation may improve outcomes in pediatric stem cell recipients.

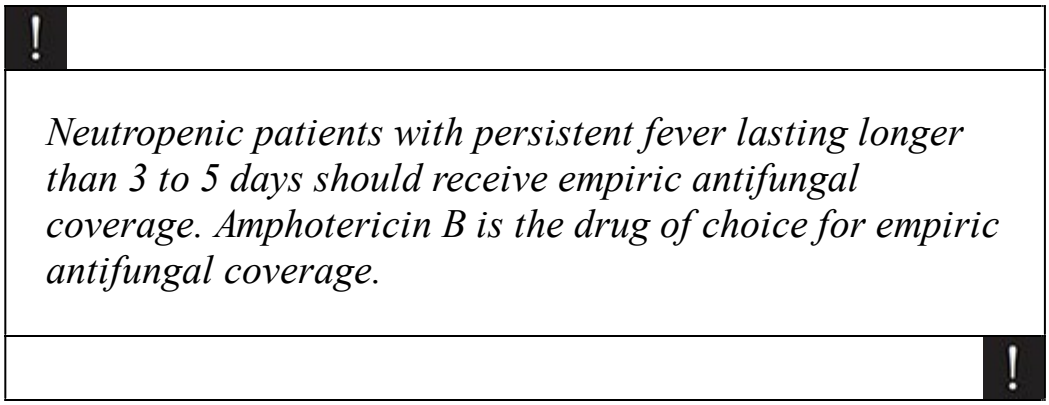
3. Neutropenic Enterocolitis (Typhlitis)

Neutropenic enterocolitis is a necrotizing inflammatory process that affects the mucosal wall of the cecum and colon. The pathogenesis is not clear, but may be related to a combination of factors, including neutropenia, mucositis, and direct cytotoxic effects of chemotherapy and, in some cases, neoplastic infiltration of the intestinal wall that creates a dysfunctional mucosa prone to microbial invasion, overgrowth, hemorrhage, and necrosis. The typical presentation of neutropenic enterocolitis mimics acute appendicitis. Most patients present with fever, abdominal pain, watery or bloody diarrhea, nausea, and vomiting. The diagnostic modality of choice is CT scan, which may demonstrate an increase in bowel wall thickness. Ultrasonography may also be useful as a bedside diagnostic tool, particularly in critically ill patients, and as a follow-up tool to assess the evolution of changes to the bowel wall. Sepsis and perforation are the most common associated complications. Early diagnosis is essential for a good outcome. The treatment of choice is conservative medical management and includes bowel rest with nasogastric decompression, platelet and red cell infusions, parenteral nutrition, and broad-spectrum antimicrobial therapy for anaerobes, Gram-negative bacteria, and fungi. Surgical interventions are indicated for patients showing clinical deterioration despite adequate medical management, abscess formation, persistent abdominal bleeding, or bowel perforation.

4. Invasive Fungal Infections

Approximately 15% to 45% of patients with persistent neutropenic fever may have invasive fungal infections. The most common are caused by *Candida albicans*, non-*albicans* species of *Candida*, and *Aspergillus fumigatus*. The increase in non-*albicans* species infections and aspergillosis is attributed to the overuse of fluconazole prophylaxis. Bronchoalveolar lavage and spinal fluid galactomannan to support pulmonary or central nervous system aspergillosis should be considered in all neutropenic patients with fever lasting ≥96 hours. Empiric antifungal therapy should be considered in patients at low risk of invasive fungal disease with prolonged fever of unclear etiology despite adequate antibiotic coverage. Patients with acute myelocytic leukemia (AML), relapsed acute leukemia, allogenic hematopoietic stem cell transplant, central venous catheter, prolonged neutropenia (>10 days), and persistently elevated C-reactive protein, and those on highly myelosuppressive

chemotherapy or steroids are at high risk of invasive fungal disease. Prophylactic screening with serum galactomannan should be considered in these patients if they are hospitalized for early detection of invasive aspergillosis. Persistent or recurrent fever of unclear etiology despite 96 hours of broad-spectrum antibiotics should be treated with empiric caspofungin or liposomal amphotericin B. A lung CT, as well as targeted imaging of other areas suspicious for infections should be considered in these patients. Additionally, a CT scan of the sinuses should be considered in children older than 2 years of age.


!
Neutropenic patients with persistent fever lasting longer than 3 to 5 days should receive empiric antifungal coverage. Amphotericin B is the drug of choice for empiric antifungal coverage.
!

Combination antifungal therapy with caspofungin and liposomal amphotericin B, followed by voriconazole maintenance treatment, has proven to be a safe regimen for immunosuppressed children with invasive pulmonary aspergillosis.

The management of infectious complications in the febrile neutropenic pediatric patient requires an aggressive multidisciplinary approach. The emergency department physician, nursing staff, respiratory therapist, hospitalist, and intensivist should work in conjunction with the pediatric oncology and infectious disease teams to develop the best management strategy for these patients.

III. SHOCK IN THE PEDIATRIC ONCOLOGY PATIENT

Case Study

A 5-year-old boy with a history of acute lymphocytic leukemia is brought into the pediatric ICU (PICU) from a referring hospital, where he presented 24 hours ago with fever, neutropenia, and thrombocytopenia. He was treated with ceftriaxone and acetaminophen (paracetamol). He became oliguric, tachycardic, and hypothermic. He received 500 mL of normal saline before transfer. On arrival to the PICU, he is found to be lethargic, with a temperature of 35.5°C (95.9°F), blood pressure of 70/30 mm Hg, and heart rate of 175 beats/min.

Detection

– What is this child's physiologic status?

– What is the most likely diagnosis?

Intervention

– Which treatment strategies take priority?

Reassessment

– Is the current treatment strategy effective?

– Does this patient need additional fluids?

– Would you consider vasopressors?

– Is endotracheal intubation indicated?

Effective Communication

– When the patient's clinical status changes, who needs the information and how will it be disseminated?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what, where, and when?

A. Risk Factors

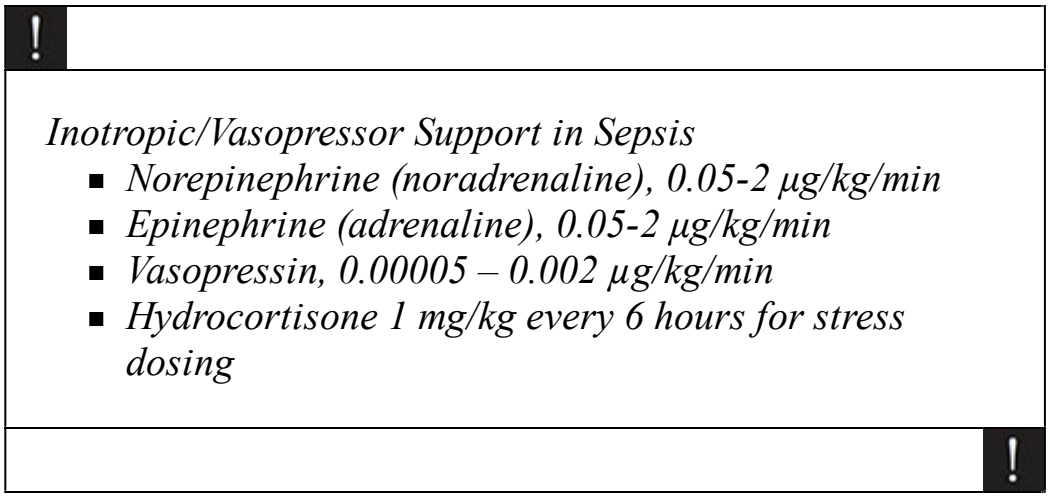
Shock and respiratory failure are the most common reasons for any child to be admitted into a PICU. Even though the clinical presentation of septic shock in oncology patients may be similar to that of noncancer patients (**Chapter 6**), the critically ill oncology patient presents additional challenges to the healthcare provider. These patients typically present with neutropenic fever, septic shock, and preexisting end-organ dysfunction.

B. Treatment

The general principles for the management and diagnosis of septic shock are covered in **Chapter 6**. The ultimate therapeutic goal in septic shock is to restore and maintain optimal organ perfusion within 1 hour. Strategies for maximizing the potential for a positive outcome include: aggressive fluid management (possibly more than 60 mL/kg in the first hour); broad-spectrum antimicrobial coverage that includes medications against Gram-positive and Gram-negative organisms, fungi, and viruses (depending on the clinical presentation); early use of inotropic and vasopressor support; and positive pressure ventilation. Early implementation of renal replacement therapy is indicated in the fluid-overloaded patient with renal insufficiency. The practitioner should be attentive to oncology patients presenting in shock but unresponsive to conventional resuscitative measures. Dilated cardiomyopathy secondary to medications (anthracyclines, high-dose cyclophosphamide, 5-fluorouracil) or radiation, pericardial effusion, and adrenal insufficiency secondary to chronic steroid use may be contributing factors to the shock state in these patients.

Corticosteroid use is indicated in patients with fluid-refractory vasopressor-resistant shock, purpura fulminans, or suspected or proven adrenocortical insufficiency. The recommended initial stress-dose of hydrocortisone dose is 1 mg/kg every 6 hours. Early transfer to a PICU for inotropic and vasopressor support and invasive cardiopulmonary monitoring is indicated in patients who do not respond to fluid resuscitation.

The care of oncology patients should be carefully directed to avoid end-organ failure because they have a substantially greater risk of mortality than do patients who do not have cancer, due to already compromised organs and immunosuppression.


Inotropic/Vasopressor Support in Sepsis
- *Norepinephrine (noradrenaline), 0.05-2 µg/kg/min*
- *Epinephrine (adrenaline), 0.05-2 µg/kg/min*
- *Vasopressin, 0.00005 – 0.002 µg/kg/min*
- *Hydrocortisone 1 mg/kg every 6 hours for stress dosing*

C. Prognosis

Although the survival rates for pediatric cancer patients with septic shock are lower than the survival rates for those who do not have cancer, recent data support an aggressive approach to care.

IV. FLUID AND ELECTROLYTE DISORDERS

Water and electrolyte disorders (**Chapter 8**) are common in the pediatric oncology patient. These disorders are usually the result of the primary tumor's impact on the kidney, the suprasellar region, or the urinary tract, or secondary to the nephrotoxic effects of chemotherapy, radiation therapy, or oncologic surgery.

Case Study

A 12-year-old previously healthy girl presents to the emergency department with fever, tachypnea, oliguria, petechiae, and generalized edema. Her initial complete blood count shows: WBC count, 250,000/mm^3; platelets, 32,000/mm^3; hemoglobin, 7.2 g/dL; lactate dehydrogenase, 3000 IU/L; uric acid, 12 mg/dL; potassium, 6.2 mEq/L (6.2 mmol/L); phosphorus, 9 mg/dL (2.9 mmol/L); and calcium, 6 mg/dL (1.5 mmol/L).

Detection

– What is this child's diagnosis?

– What is she at risk of?

Intervention

– Which treatment strategies take priority?

– Would an exchange transfusion be appropriate?

– Should her hypocalcemia be treated?

Reassessment

– Is the current treatment strategy effective?

– Does she need additional fluids?

– What types of fluids are indicated?

– Is a blood transfusion needed?

– Is a platelet infusion indicated?

Effective Communication

– Do you need to contact the nephrologist?

– Where is the best place to manage the care of this patient?

Teamwork

– Who is to do what, where, and when?

A. Tumor Lysis Syndrome

Tumor lysis syndrome (TLS) is a life-threatening complication resulting from massive and rapid tumor cell lysis with the release of intracellular metabolites, including nucleic acids, urea nitrogen, phosphorus, and potassium, leading to hyperuricemia, hyperphosphatemia, hyperkalemia, and secondary hypocalcemia. TLS most often develops 12 to 72 hours after the initiation of chemotherapy but may also occur spontaneously or after administration of steroids, hormones, or radiation therapy.

1. Diagnosis

TLS most frequently occurs in patients with a large tumor burden (Burkitt lymphoma, T-cell acute lymphocytic leukemia, acute lymphocytic leukemia or AML with WBC >100,000 mm^3), or in instances where a solid tumor is widely disseminated, rapidly proliferating, or highly sensitive to chemotherapy. The 2004 Cairo and Bishop Classification system defines 2 TLS categories and a risk

assignment based on tumor type, staging, and laboratory values (low risk, ≤1%; intermediate risk, 1%-5%; high risk, ≥5%).

a. Laboratory Tumor Lysis Syndrome

This TLS is most common and is characterized by the occurrence of ≥2 of the following values within 3 days before and up to 7 days after initiation of cancer treatment:

- Uric acid: increase of 25% from baseline or value ≥8 mg/dL (>475.8 μmol/L)
- Potassium: increase of 25% from baseline or value ≥6 mEq/L (>6 mmol/L)
- Phosphorus: increase of 25% from baseline or value ≥6.5 mg/dL (>2.10 mmol/L)
- Calcium: decrease of 25% from baseline or value ≤7 mg/dL (<1.75 mmol/L)

b. Clinical Tumor Lysis Syndrome

This is determined by the presence of laboratory TLS and at least 1 of the following clinical manifestations:

- Renal failure (estimated glomerular filtration rate of ≤60 mL/min and/or serum creatinine 1.5 times higher than the upper normal limit for age)
- Cardiac arrhythmia
- Seizures

2. Symptoms

The symptoms most commonly found in patients with TLS include: anorexia, malaise, weakness, vomiting, hiccups, paresthesia, tetany, carpopedal spasm, oliguria, anuria, lethargy, encephalopathy, seizures, electrocardiogram changes with high peaked T waves to life-threatening arrhythmias, syncope, shock, and death if uncorrected.

3. Pathophysiology

The high cell turnover with the release from cellular breakdown, accentuated by chemotherapy, leads to a dramatic rise in plasma levels of uric acid, urea nitrogen, phosphorus, and potassium that rapidly saturates renal homeostatic mechanisms. The glomerular filtration rate declines and progresses to renal failure, as a result of the precipitation of calcium phosphate and uric acid crystals in the renal tubules. Patients with dehydration, preexisting renal disease, or obstruction to urinary flow from tumor involvement are at higher risk of developing TLS.

4. Treatment

The key to the management of TLS is early diagnosis of the primary condition, rapid recognition of the risk factors, and prompt initiation of preventive treatment. A TLS treatment summary is presented

in **Table 17-3**.

Table 17-3	TREATMENT OF TUMOR LYSIS SYNDROME

- Hydrate aggressively with 5% dextrose in normal saline at a rate of 3000 mL/m²/d or 200 mL/kg/d if <10 kg.
- Control uric acid:
  – Rasburicase: 0.15-0.2 mg/kg in a 30-min IV infusion, repeated daily if needed. Contraindicated in patients with glucose-6-phosphate dehydrogenase deficiency and methemoglobinemia.
  OR
  – Allopurinol: 100 mg/m² orally every 8 h, to be given 24-48 h before initiation of chemotherapy.
- Monitor urine output continuously (goal >100 mL/m² or >4-6 mL/kg/h). Consider cautious use of diuretics.
- Maintain specific gravity ~1.010.
- Restrict potassium and phosphorus; use cardiopulmonary monitoring in hyperkalemic patients.
- Replace calcium only in symptomatic patients (positive Chvostek or Trousseau sign or electrocardiographic changes)
- Consider early renal replacement therapy in patients with refractory hyperkalemia or symptomatic hyperphosphatemia, or with rapid decline in renal function.

Essential treatment measures are as follows.

a. Monitoring

Patients with TLS, or at risk to develop TLS, should be monitored for electrocardiographic changes associated with hyperkalemia (prolonged PR interval, flattened P wave, widened QRS complex, and peaked T wave) and/or hypocalcemia (prolonged QT interval). Levels of sodium, potassium, phosphorus, calcium, magnesium, creatinine and urea nitrogen, lactate dehydrogenase, and uric acid, should be monitored every 4 to 6 hours for the first 48 hours, then every 12 hours for the next 3 days, and once daily subsequently.

b. Hydration

IV hydration should begin immediately and continue for at least 48 hours after specific tumor therapy is initiated to improve renal blood flow and glomerular filtration. The infusion rate should be 3000 mL/m²/d or 200 mL/kg/d if patient weight is <10 kg. Urine output should be continuously monitored and kept at ≥100 mL/m²/h (4-6 mL/kg/h). Furosemide or mannitol may be required to maintain this urinary output, but such use is discouraged in the volume-depleted patient or in those with obstructive uropathy.

c. Urine Alkalinization

Urine alkalinization is no longer recommended. It may improve uric acid solubility (maximum solubility at pH 7.5), but it does not increase the solubility of xanthine and hypoxanthine metabolites. In addition, and perhaps more relevant, urine alkalinization decreases the excretion of phosphorus and may lead to calcium-phosphate precipitation in the renal tubules.

d. Uric Acid Control

Allopurinol should be administered orally at a dose of 100 mg/m² thrice daily concomitantly with IV hydration and diuresis in low-risk patients. Allopurinol prevents new uric acid formation but will not

affect circulating uric acid levels. It may take 2 to 4 days for uric acid level normalization. Allopurinol may potentiate the effect of agents like 6-mercaptopurine and azathioprine, as well as cause additional bone marrow suppression when used with cyclophosphamide.

Rasburicase should be administered at a dose of 0.15-0.2 mg/kg IV given over 30 minutes in high-risk conditions. The first dose should be administered at least 4 hours before tumor-specific therapy and can be continued as needed for up to 7 days. Concomitant use of both allopurinol and rasburicase is not recommended. Rasburicase promotes the metabolism of uric acid into a soluble compound, allantoin. Rasburicase is degraded by peptide hydrolysis and does not impact renal or hepatic function, or affect cytochrome P-450. It is contraindicated in patients with glucose-6-phosphate dehydrogenase deficiency and a history of methemoglobinemia. Rasburicase is the drug of choice for at-risk patients and may be considered in a single dose for those at intermediate risk. Recent studies have shown a shorter ICU stay, shorter overall hospital stay, and lower total inpatient cost in patients treated with rasburicase compared to those receiving allopurinol.

e. Hyperkalemia and Hyperphosphatemia

Potassium and phosphorus replacements should be discontinued and intake restricted.

f. Management of Severe Electrolyte Disturbances

Patients with symptomatic hyperkalemia (>6 mEq/L [>6 mmol/L]) or with electro-cardiographic signs of hyperkalemia should receive calcium, insulin/glucose, bicarbonate, and a β-agonist (**Chapter 8**). Diuretics should be used with caution in dehydrated patients. In the presence of asymptomatic hyperkalemia, sodium polystyrene sulfonate (Kayexalate), taken orally at 1 g/kg, may be helpful in preventing symptomatic hyperkalemia and potassium rebound after the transient effect of acute hyperkalemia treatment.

Patients with hyperphosphatemia should receive oral phosphate binders, such as sevelamer hydrochloride or aluminum hydroxide.

Calcium replacement with a single dose of calcium gluconate, 50 mg/kg, is indicated during TLS only in patients with neuromuscular irritability secondary to hypocalcemia, including seizures, arrhythmias, or positive Chvostek or Trousseau sign. The treatment of asymptomatic hypocalcemia is contraindicated.

Renal replacement therapy (hemodialysis, continuous venovenous hemofiltration) should be initiated in patients with refractory hyperkalemia, severe metabolic acidosis, volume overload unresponsive to diuretics, hyperphosphatemia not responding to medical treatment, and uremic symptoms (including pericarditis and encephalopathy), or in those with rapidly declining renal function.

V. NEUROLOGIC EMERGENCIES

A. Hyperleukocytosis

Hyperleukocytosis is defined as a WBC count ≥100,000/mm³ in the peripheral blood.

1. Risk Factors

Hyperleukocytosis occurs in children with AML, acute lymphocytic leukemia, chronic myelogenous leukemia, and myeloproliferative disorders. Children with hyperleukocytosis are at increased risk for pulmonary and central nervous system (CNS) complications due to leukostasis (slugging of leukemic cells in capillary vessels). Children with AML are at the greatest risk because monocyte blasts are more adherent to vessel walls. Patients with hyperleukocytosis are at risk for TLS, cerebral stroke, and pulmonary stasis.

2. Clinical Findings

Clinically significant hyperleukocytosis occurs with a WBC count of ≥200,000/mm³ in patients with AML and with a count in excess of 300,000/mm³ in patients with acute lymphocytic leukemia and chronic myelogenous leukemia. Most patients are asymptomatic at presentation, but some may present with subtle CNS signs, such as headaches, tinnitus, dizziness, and blurred vision. The presentation sometimes is more dramatic and includes full-blown symptoms of cerebral stroke (changes in mental status, focalization, seizures, and increased intracranial pressure). Pulmonary manifestations include tachypnea, dyspnea, hypoxia, and acidosis.

Children with hyperleukocytosis often develop a consumptive coagulopathy, likely through activation of the extrinsic pathway. The platelet count, prothrombin time, partial thromboplastin time, and fibrinogen level should be monitored frequently. CT and/or magnetic resonance imaging, with and without contrast, can be useful in evaluating the nature and extent of a stroke.

3. Treatment

The treatment of hyperleukocytosis aims to prevent TLS and reduce tumor burden through hydration, induction chemotherapy, leukapheresis, or exchange transfusion. The management of stroke and pulmonary leukostasis is supportive. Treating the underlying malignancy may prevent additional strokes and help decrease further pulmonary involvement. Platelets should be maintained at levels greater than 30,000 to 50,000/mm³. Coagulation disorders should be corrected promptly. Packed red blood cell (PRBC) transfusions are discouraged in patients with asymptomatic anemia because of the risk of worsening hyperviscosity. Platelet transfusions do not increase viscosity of blood.

B. Spinal Cord Compression

Case Study

A 15-year-old high school basketball player presents to the emergency department with a 1-month history of progressive back pain radiating to the right lower extremity, which was initially relieved by ibuprofen. The patient now complains of right lower-extremity weakness and paresthesias.

Physical examination reveals tenderness to percussion on the back, decreased strength and hyporeflexia of the right lower extremity, and poor sphincter tone on rectal examination.

Detection

– What is the possible diagnosis?

Intervention

– What diagnostic modality is indicated?

– Which pharmacologic treatment should be initiated?

– Should the radiation therapy or neurosurgery department be contacted?

Reassessment

– Is the current treatment strategy effective?

Effective Communication

– Who needs to know if the patient continues to have neurologic deterioration?

– Where is the best place to manage this patient?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

1. Pathophysiology

Spinal cord compression in children with cancer most frequently results from metastatic spread (85%) of the tumor rather than primary disease. The most common metastatic tumors involving the spinal cord are sarcomas (Ewing sarcoma, osteosarcoma), neuroblastomas, lymphomas, germ cell tumors, and metastatic CNS neoplasms. Tumors typically infiltrate by local extension into the epidural space through the intervertebral foramina from nearby structures or by hematogenous spread. Most tumors are primarily located in the lumbosacral region and surround the spinal cord. The spinal cord and the vertebral venous plexus become progressively compressed by the encircling tumor, resulting in vasogenic cord edema, hemorrhage, and ischemia.

2. Symptoms

Pain, either local or radicular, is the most frequently encountered presenting sign of spinal cord compression in children (80%). The onset of pain is often insidious and follows a gradually progressive course (weeks to months). Pain associated with epidural compression of the spinal cord is exacerbated when the patient is recumbent and improves when the patient is in an upright position. Once motor weakness and sensory deficits appear, disease progression may be rapid. Autonomic

dysfunction (bladder or bowel incontinence) is a late and ominous sign of epidural compression. Lesions above T10 produce hyperreflexia and a positive Babinski reflex, whereas lesions below T10 that compress the conus or cauda equina produce hyporeflexia.

3. Diagnosis

MRI with gadolinium enhancement of the entire spine is the recommended diagnostic modality for children with suspected spinal cord involvement. Craniospinal T1-weighted and T2-weighted MRI allows demonstration of epidural involvement, intraparenchymal spread, and compression of nerve roots. Conventional radiographs may reveal bone destruction but do not provide information about the spinal cord structure. Lumbar puncture is relatively contraindicated in spinal cord tumors except in leptomeningeal disease.

4. Treatment

Early diagnosis and treatment is vital for a child with cancer and confirmed spinal cord compression. High-dose steroids (dexamethasone, 1-2 mg/kg) should be administered immediately, followed by lower doses (0.25-0.5 mg/kg IV every 6 hours) once the diagnosis is confirmed. Emergent laminectomy with posterior decompression, radiation therapy, and chemotherapy should be initiated as indicated. Non-oncologic diagnoses must be excluded (tuberculosis, osteomyelitis). Prompt referral to a pediatric hospital for pediatric neurosurgery and a pediatric oncology evaluation is warranted. The prognosis for recovery in those with neurologic dysfunction is related to the severity and duration of symptoms at the time of diagnosis. About 66% of children treated regain some motor and sensory function.

Spinal Cord Compression:
Diagnosis and Treatment

- *Emergent MRI with gadolinium*
- *Dexamethasone: 1-2 mg/kg IV first dose, then 0.25-0.5 mg/kg every 6 hours*
- *Emergent laminectomy and posterior decompression*
- *Radiation and chemotherapy*

VI. HEMATOLOGY: COAGULATION AND TRANSFUSIONS

Case Study

An 8-year-old, African-American girl undergoing chemotherapy for acute lymphoblastic leukemia is in the clinic to receive her regular (every 3-4 weeks) red blood cell transfusion. After 30 minutes of the transfusion, she develops chills, fever, hypotension, gross hematuria, and extensive bleeding from her mucosal membranes. Her initial laboratory results show: WBC count, 32,000 x 10^9 cells/L (68% neutrophils, 20% bands, 12% lymphocytes); hemoglobin, 4.5 g/dL; platelet count, 54,000 x 10^9 cells/L; prothrombin time (PT), 38 seconds; activated partial thromboplastin time (aPTT), 58 seconds; fibrinogen, 50 mg/dL (1.47 µmol/L); and D-dimer >10,000 ng/mL (normal, 0-230 ng/mL).

Detection

– What is the most likely diagnosis?

Intervention

– Which treatment strategies take priority?

– What diagnostic modality is indicated?

– Is a blood transfusion indicated?

Reassessment

– Is the current treatment strategy effective?

Effective Communication

– When the patient's clinical status changes, who needs this information and how will it be disseminated?

– Should the patient be transferred to the PICU?

Teamwork

– How will the treatment strategy be implemented?

– Who is to do what and when?

In this particular case, the transfusion needs to be stopped immediately, critical care support must be given, and depleted coagulation factors and red blood cells should be replaced with fresh frozen plasma (FFP), cryoprecipitate, and PRBCs. Empiric antibiotics should be administered to treat possible transfusion-associated bacterial infection.

Hemostasis is maintained through a delicate balance between procoagulant and antithrombotic factors. Severe deficiency of coagulation proteins can lead to bleeding or clotting. Clinically significant decreases in red blood cells, platelets, or coagulation factors require therapeutic intervention. Transfusion of blood products can assist in oxygen-carrying capacity, expanding the vascular volume, and improving coagulation and hemostasis. PRBCs are the most common blood component used, followed by platelets and FFP.

Transfusion of blood products can assist in an oxygen-carrying capacity, expanding the vascular volume and improving coagulation and hemostasis.

A. Coagulopathies

Disseminated intravascular coagulation (DIC) is an acquired syndrome characterized by diffuse activation of the coagulation system by various causes, leading to fibrin deposition and clotting inside the vascular compartment. Resulting diffuse microthrombi cause endothelial injury, end-organ dysfunction, ischemia to extremities, and skin necrosis. Damage to the red blood cell membrane while passing through damaged vasculature causes intravascular hemolysis. Thrombosis in the microvasculature results in a significant decrease in platelets and coagulation factors, leading to bleeding tendency; this presents as purpura fulminans, mucosal bleeding, petechial hemorrhages, bleeding from central and peripheral lines, and possibly intracranial bleeding. The main causes of DIC in children are infection (eg, meningococcemia), malignancy, massive tissue damage, and immunologic reaction.

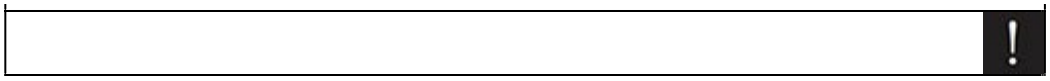
DIC: Diagnosis and Treatment

- *Treat the underlying cause.*
- *Replace necessary factors.*

The diagnosis of DIC is not made based on a specific test, but rather when the proper laboratory evidence and clinical findings are present. Common laboratory findings consists of an elevated PT and aPTT, decreased fibrinogen, elevated D-dimer, decreased platelet count, decreased concentration of factors V and VIII, and fragmented red blood cells on the peripheral smear. These abnormalities are a reflection of the excessive thrombosis, resulting in the consumption of platelets, coagulation factors, and fibrinolysis.

Differential Diagnosis of Acquired Coagulopathies

- *Vitamin K deficiency*
- *Liver disease*
- *Renal disease*
- *Drug-induced coagulopathy*



The most effective therapy begins with the treatment of the underlying illness, thus eliminating the catalysts of intravascular coagulation. Replacing deficient coagulation factors and platelets should be reserved for patients with active bleeding. The replacement should begin with platelets and FFP. Transfusion of cryoprecipitate is required for the replacement of fibrinogen. Follow-up laboratory studies are necessary to monitor the correction of deficiencies.

B. Blood Products

The transfusion of blood products in patients newly diagnosed with a malignancy is frequently necessary in the critical care setting, but it is not without risk or complication. These complications arise from both infectious and noninfectious origins. Over the past 30 years, significant advances in the screening of blood products and utilization of pathogen reduction treatments have led to a decrease in the incidence of infections. Still, the possibility continues and must be carefully weighed. The decision to transfuse a patient should only be made when it is clinically indicated. Informed consent should always be obtained before the administration of blood products.

To decrease the chance of clerical error, all blood products drawn for typing and crossmatching must be clearly labeled with the patient's identification information. Before initiating the transfusion, the patient's identification band must be checked and blood type verified. Administration of incompatible blood products can result in serious consequences, including death.

1. Red Blood Cells

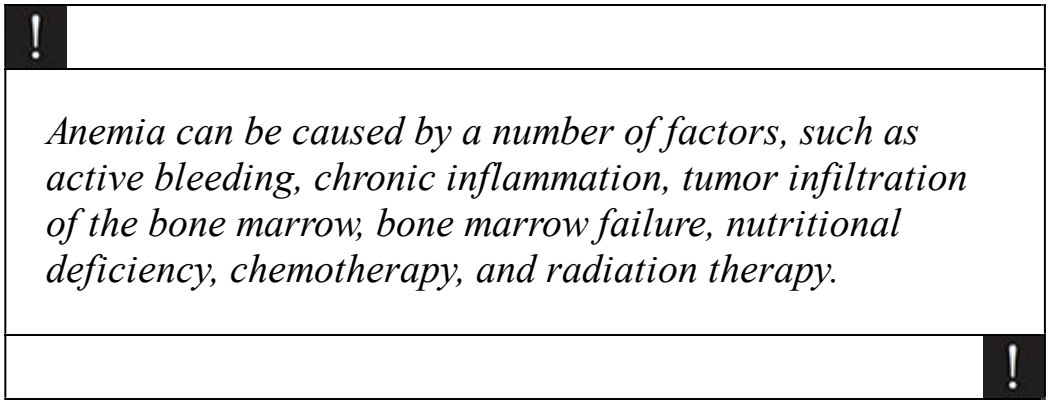
Anemia is frequently encountered in the critical care setting. Red blood cells should only be administered with the consideration of clinical signs and symptoms (**Table 17-4**). A sudden loss of blood can compromise the cardiovascular system. Healthy, young individuals can tolerate a loss of approximately 30% (~16 mL/kg) of their blood volume. Unless rapid intervention occurs, patients who lose 50% of their blood volume proceed to heart failure and death.

Table 17-4	CLINICAL SIGNS AND SYMPTOMS OF ANEMIA
General: Patients with sudden loss of blood can present with confusion and decreased responsiveness, as seen in those with hypovolemic shock. Patients with a gradual onset of anemia have decreased stamina, headache, irritability, and anorexia. **Skin color:** Check for pallor in the normally pink mucosa and nail beds, mottling, cool clammy skin, and delayed capillary refill. Prolonged iron deficiency can lead to softening and spooning of the fingernails. **Tachypnea:** Rapid breathing and air hunger are important signs of anemia in infants and young children. Healthy adolescents tolerate moderately severe anemia with only dyspnea associated with exercise. **Tachycardia:** Patients with hypovolemic shock will have a rapid and thready pulse; those with prolonged anemia have an audible flow murmur on auscultation.	

Patients with slow blood loss or chronic anemia are usually well-compensated. Subsequently, they may lack symptoms and not seek medical attention immediately, but they may quickly decompensate if otherwise stressed. These patients often present with clinical signs and symptoms of anemia that include headache, fatigue, dyspnea, irritability, anorexia, and tachycardia.

The underlying reason for anemia should be determined and, when possible, corrected to avoid additional transfusions. Anemia can be caused by a number of factors, such as active bleeding, chronic inflammation, tumor infiltration of the bone marrow, bone marrow failure, nutritional deficiency, chemotherapy, and radiation therapy. The transfusion of PRBCs should be considered when the hemoglobin level falls below 7 g/dL. However, the decision to transfuse should be based on clinical parameters and context, not solely on a laboratory value. The volume of PRBCs transfused in small pediatric patients who require less than 1 U should be based on body weight. As a general rule, 5 to 10 mL/kg of PRBCs will increase the recipient's hemoglobin by 2 to 4 g/dL. The standard dose of PRBCs is 10 to 15 mL/kg administered over 2 to 4 hours to avoid congestive heart failure. Rapid replacement of PRBCs is indicated in cases of acute blood loss or shock.

Severely anemic patients scheduled to undergo a surgical procedure or radiation therapy should be transfused prophylactically. They should not be rapidly transfused, because congestive heart failure may be induced; it is preferable to transfuse the severely anemic patient in smaller aliquots. Consider transfusion in patients with a hemoglobin <5 g/dL, using the rate of 1 mL x hemoglobin (g)/kg/h if there is evidence of cardiovascular instability. One unit of PRBCs may contain 225 to 350 mL and, depending on the blood bank, may have a hematocrit of 55% to 80%. PRBCs are the product of choice to restore oxygen-carrying capacity and red cell mass. PRBCs must be ABO and Rh(D) compatible to prevent a massive transfusion reaction. Crossmatching is required to prevent transfusion of incompatible minor blood-group antigens. Only in a life-threatening emergency should O-negative products be utilized.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Anemia can be caused by a number of factors, such as active bleeding, chronic inflammation, tumor infiltration of the bone marrow, bone marrow failure, nutritional deficiency, chemotherapy, and radiation therapy.
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The use of leukocyte filters has become standard practice in many institutions. Leukocyte-depleted red blood cells reduce the incidence of febrile reactions and other cytokine-induced adverse events. The use of leukocyte-depleted red blood cells reduces alloimmunization to donor HLA antigens, which is greatly beneficial to patients awaiting an organ transplant. These filters remove 99.9% of the leukocytes, which cause fever. Additionally, washed PRBCs effectively remove the unwanted cells, while maintaining approximately 85% of the red cell mass. The use of irradiated blood is standard practice in cancer centers but is normally reserved for patients likely to undergo a bone marrow transplant.

There is a high incidence of inherited defects in the globin structure in patients of African, Indian, Asian, and Mediterranean populations. The homozygous form of sickle cell anemia is a debilitating disease. Early in life, these patients present with severe hemolytic anemia and vaso-occlusive disease. Patients with sickle cell anemia are at an increased risk of death secondary to sepsis or CNS thrombosis. Patients with hemoglobinopathies, such as sickle cell disease, are at increased risk for

vaso-occlusive infarction of marrow, the spleen, and renal medullary tissue. They often have recurrent painful crises.

Hypertransfusion therapy of PRBCs and maintaining the hemoglobin levels above 10 g/dL decreases production of sickle red blood cells. This significantly decreases morbidity, the risk of other life-threatening events, and the frequency of painful crises. Patients with sickle cell disease have an increased risk of stroke and hyperviscosity, and, therefore, should not be transfused to a hemoglobin level above 12 g/dL.

Hypertransfusion is also effective in minimizing the production of hemoglobin S. Because of the inherent risks, prophylactic transfusions are reserved for patients who are severely ill, and recommended for patients with a history of CNS thrombosis or transcranial Doppler blood flow velocities >200 cm/s in the middle cerebral or internal carotid arteries.

The use of exchange transfusion in sickle cell patients is also effective in reducing the sickle cells. This modality is beneficial to patients with recurrent stroke, because there is no increase in hematocrit or blood viscosity. Exchange transfusions are also indicated in patients with hyperleukocytosis or hyperbilirubinemia.

Pediatric oncology patients commonly are anemic at diagnosis. In addition, they may be significantly anemic while undergoing chemotherapy and are routinely transfused with PRBCs. They are usually transfused if the hemoglobin level is <8 g/dL and to maintain a level above 10 g/dL when receiving radiation therapy. Premature neonates, patients with certain congenital immunodeficiencies (such as patients with DiGeorge syndrome), and those undergoing chemotherapy should receive irradiated blood products to prevent transfusion-associated graft-versus-host disease.

The dose of PRBCs is dependent on the desired hemoglobin or hematocrit level, the rate of red cell destruction, and the presence of blood loss. Patients with severe anemia, but no ongoing blood loss, who receive 10 to 15 mL/kg should have hemoglobin increased by 2.5 to 3 g/dL and the hematocrit by 10%. Indications for red blood cell transfusion are listed in **Table 17-5**.

Table 17-5 INDICATIONS FOR RED BLOOD CELL TRANSFUSION

1. Infants <4 months of age
    a. Hgb level <13 g/dL in neonates <24 h old
    b. Hgb level <13 g/dL and presence of severe pulmonary disease, cyanotic heart disease, or heart failure
    c. Acute blood loss of at least 10% of total blood volume
    d. Phlebotomy losses of at least 10% of the total blood volume
    e. Hgb level <9 g/dL in infants with clinical manifestations of anemia
2. Children >4 months of age
    a. Hgb level <7 g/dL
    b. Hgb level <8 g/dL in patients undergoing surgery or invasive procedures
    c. Intraoperative blood loss of 10% to 15% or more of total blood volume, postoperative Hgb level <8 g/dL, and signs or symptoms of anemia
    d. Hgb level <9 g/dL in patients with symptomatic anemia
    e. Hgb level <10 g/dL in patients receiving radiation therapy
    f. Acute blood loss with hypovolemia unresponsive to crystalloid or colloid

2. Platelets

Platelets have an important role in hemostasis and coagulation. An adequate number of functional platelets are required for hemostasis. After vascular injury, the formation of the primary hemostatic plug prevents the flow of blood from small- and medium-sized blood vessels. In addition, platelets release thromboxane A_2, which mediates the change in vascular tone and platelet activation.

A healthy individual with functional platelets can withstand a platelet count as low as 10,000/mm³. Individuals with fever or infection or who are acutely ill have a much higher risk of bleeding, even with platelet counts as high as 30,000/mm³.

Clinical signs and symptoms of a reduced platelet count, known as *thrombocytopenia*, are nonspecific but primarily consist of gingival bleeding, epistaxis, hematuria, and petechial rash. The rash is most commonly seen on the upper and lower extremities. Diagnosis of thrombocytopenia is based upon the platelet count.

Platelet transfusion is used to prevent bleeding and to treat bleeding secondary to thrombocytopenia or platelet dysfunction. It is not indicated for patients with rapid platelet destruction problems, such as immune thrombocytopenic purpura; in this circumstance, a transfusion is only indicated with life-threatening hemorrhage. Platelet transfusions are useful in the bleeding patient with a consumptive coagulopathy, such as DIC. Platelets are frequently administered to patients receiving chemotherapy or who have bone marrow infiltration. Indications for platelet transfusions are listed in **Table 17-6**.

Table 17-6	INDICATIONS FOR PLATELET TRANSFUSIONS

Premature or sick infants

- Platelet count <50,000 /mm³
- Platelet count <100,000/mm³ for hemodynamically unstable infants

Children

- Platelet count <10,000/mm³ with evidence of failure of platelet production
- Active bleeding with a qualitative platelet defect
- Platelet count <30,000/mm³ with active bleeding
- Platelet count <50,000/mm³ with an anticipated minor invasive procedure, <100,000/mm³ with an anticipated major surgery

Oncology patients

- Platelet count <10,000/mm³ without active bleeding and failure of platelet production
- Platelet count <10,000/mm³ when undergoing induction chemotherapy for acute lymphocytic leukemia
- Platelet count <20,000/mm³ when undergoing induction chemotherapy for acute myelogenous leukemia
- Platelet count <30,000/mm³ for patients with tumors in the central nervous system
- Platelet count <20,000/mm³ before lumbar puncture
- Platelet count <100,000/mm³ in patients with a high risk of traumatic taps
- Platelet count <50,000/mm³ in a bleeding patient with normal coagulation studies
- Platelet count <20,000/mm³ before an intramuscular injection

One unit of random platelets per each 10 kg body weight, will increase the platelet count by approximately 50,000/mm³ if there is no active consumptive process (fever, immune thrombocytopenic purpura, sepsis, alloimmunization, DIC) or sequestration. The platelet count should be checked 1 to 2 hours posttransfusion to identify refractory patients.

If the platelet count does not increase as expected following 2 separate transfusions, the patient may

have immune-mediated platelet refractoriness secondary to alloantibodies. Nonimmune causes of this condition are common and include splenomegaly, fever, infection, DIC, and the use of amphotericin.

For refractory patients, a trial of HLA-matched platelets is indicated. Should this fail, other treatment possibilities include leukocyte-depleted HLA-matched platelets or massive transfusion with random donor platelets, the latter of which will saturate the antibody concentration.

Pheresis platelets are harvested from a single donor and generally contain >30,000/mm³ platelets, which is equal to 6 to 8 units of random donor platelets. The volume is usually 250 to 300 mL. The use of platelets from single-donor apheresis is indicated for patients who are frequently transfused or need multiple units to decrease donor exposure and the inherent risks.

3. Plasma Derivatives

a. Fresh Frozen Plasma

Patients with severe clotting deficiencies or with a consumptive coagulopathy may require derivatives of plasma. FFP is a rich source of clotting factors, containing a high concentration of fibrinogen, factor VIII, von Willebrand factor, and factor XIII. It is frequently used in patients unable to synthesize clotting factors due to liver disease or in those with a consumptive coagulopathy secondary to trauma.

FFP is plasma that has been harvested from whole blood. Whole blood is centrifuged, and the excess plasma is then frozen within 8 hours of collection. FFP can be stored for 1 year at -20°C (-4°F). It is thawed at 37°C (98.6°F) for 30 to 60 minutes before transfusion. To increase the likelihood of the product remaining sterile and containing adequate levels of coagulation factors, FFP must be administered within 8 hours of thawing. Additionally, the product must be ABO compatible with the patient's blood type.

Patients with a significantly prolonged PT and aPTT have 15% to 25% of their normal factor levels. Large amounts of FFP are required to increase the coagulation factor levels above 30%. Each unit contains about 250 mL of plasma and yields only a 3% increase in coagulation factor levels. Indications for transfusion of FFP are listed in **Table 17-7**.

Table 17-7	INDICATIONS FOR TRANSFUSION OF FRESH FROZEN PLASMA

- Emergency reversal of warfarin anticoagulation
- Disseminated intravascular coagulation
- Thrombotic thrombocytopenic purpura
- Prolonged partial thromboplastin time >60 s
- Prolonged prothrombin time >18 s despite administration of vitamin K
- Liver disease
- Massive blood loss

FFP is administered at 10 to 20 mL/kg and should be infused at a maximum rate of 0.5 mL/kg/min. The use of FFP should be reserved for situations in which a concentrate of coagulation factors is not necessary or not available. Generally FFP is no longer used for administration of factor VIII or IX.

b. Cryoprecipitate

The preparation of cryoprecipitate consists of flash freezing fresh plasma. It is then thawed at 4°C (39.2°F). The residual precipitate contains 150 to 200 mg of fibrinogen per 15 mL. Cryoprecipitate also contains factor VIII, factor XIII, von Willebrand factor, and fibronectin at much higher concentrations than in FFP. The use of cryoprecipitate is indicated when fibrinogen levels are <100 mg/dL. It should be transfused within 4 hours of thawing.

Cryoprecipitate dosing is 1 unit per 5 to 10 kg. One unit contains 10 to 15 mL of volume and will raise the fibrinogen level by 5 to 10 mg/dL per unit. With proper dosing, the expected rise should be 60 to 100 mg/dL. The therapeutic goal is a fibrinogen level >100 mg/dL.

C. Adverse Reactions

The main cause of acute hemolytic reactions is ABO incompatibility. Treatment seeks to eliminate the precipitating agent and provide general supportive care. The incidence of adverse reactions associated with the transfusion of blood products is about 5%. Most are mild urticarial reactions that can be treated by stopping the transfusion for 30 minutes, followed by the administration of antihistamines. If the symptoms subside, the transfusion can be restarted. All other reactions require discontinuation of the transfusion, and the transfused product and a patient blood sample must be sent to the blood bank for further evaluation.

The medical treatment of transfusion reactions is detailed in **Table 17-8**.

Table 17-8 TREATMENT OF TRANSFUSION REACTIONS

Medication	Dosage	Indications
Diphenhydramine	0.5-1 mg/kg IV (maximum dose, 50 mg)	Urticaria and hives
Acetaminophen/paracetamol	10-15 mg/kg PO (maximum dose, 1000 mg)	Fever and chills
Hydrocortisone	1-2 mg/kg IV (maximum dose, 100 mg)	Severe urticaria, diaphoresis, rigor, or pallor
Epinephrine/adrenaline (1:1000)	0.01 mL/kg IM (maximum dose, 0.5 mL/dose)	Shock[a], bronchospasm, and hypotension
Furosemide	1-2 mg/kg IV (infants) 0.5-1 mg/kg (older children and adolescents; maximum dose, 40 mg)	Acute hemolytic reaction – maintain urine flow >1 mL/kg/h
Oxygen	100% via non-rebreather face mask	

PO, orally; IM, intramuscular

[a]Patients with shock may require additional therapeutic interventions, including fluids, advanced airway management protection, and vasoactive medications.

1. Allergic Reactions

Severe allergic reactions can present with urticaria followed by laryngospasm, bronchospasm, and angioedema. These patients require immediate cessation of the transfusion, followed by administration of steroids or possibly epinephrine. Close observation is needed for 4 to 6 hours if the patient's reaction is mild. High-flow oxygen, maintenance of an open airway, close cardiac monitoring, and IV access should be considered in asymptomatic patients with a history of serious anaphylactic reaction. Anaphylaxis, an immunoglobulin E-mediated allergic reaction, is a rare complication, but it can result in death if not treated immediately. Most anaphylactoid reactions (resembling anaphylaxis but caused by a nonimmunologic mechanism) occur in patients with immunoglobulin A deficiency. The transfusion should not be restarted after any severe reaction.

2. Febrile Nonhemolytic Reactions

Even after washing or filtering PRBCs, a percentage of leukocytes and cytokines remain. The presence of leukocytes results in the release of cytokines during transfusion in patients with antibodies that react with alloantigens in the blood product. This often results in fever, chills, or diaphoresis. These symptoms can also occur if the blood product is contaminated with bacteria. Individuals receiving transfusions over an extended period should be given leukocyte-depleted blood products to help minimize the development of antibodies that react with foreign WBCs.

The treatment of febrile nonhemolytic reactions is cessation of the transfusion. The patient should be examined, and the vital signs should be monitored. Symptoms should be treated with antipyretics and possibly antibiotics. The transfused product should be evaluated by culture and repeated crossmatch.

3. Acute Hemolytic Transfusion Reactions

Transfusion of ABO-incompatible blood products results in acute hemolytic transfusion reactions. This is usually a result of mislabeled blood products or the administration of blood products to the wrong patient, resulting in the immunologic destruction of the transfused red cells.

Clinical signs and symptoms of acute hemolytic reactions consist of fever, chills, urticaria, dyspnea, tachycardia, nausea, abdominal or lower back pain, hyper- or hypotension, jaundice, hemoglobinuria (port wine-colored urine), and shock. The hemoglobinuria leads to oliguria and, eventually, renal failure. Laboratory evaluation should include a complete blood count, coagulation studies, urinalysis, and a direct Coombs test. Significant laboratory findings may include anemia, DIC, hemoglobinuria, and a positive result on the Coombs test.

Therapy for acute hemolytic transfusion reactions involves immediate cessation of the transfusion, fluid support to maintain adequate hemodynamic status, and diuretics (mannitol) if needed to maintain adequate renal blood flow. The urine output should approximate 1 to 2 mL/kg/h. Vasopressors and inotropes may be required for hemodynamic stability. The use of steroids is indicated in the management of acute hemolytic reaction transfusions because of the effectiveness in suppressing the inflammatory reaction.

4. Delayed Hemolytic Transfusion Reactions

There are two different types of delayed hemolytic transfusion reactions: primary immunization and anamnestic response. Primary immunization is characterized as a mild reaction tending to occur

weeks after the administration of blood products. This type of reaction rarely leads to significant hemolysis. Laboratory evaluation will usually demonstrate a decrease in the hemoglobin level 2 to 4 weeks after transfusion.

The anamnestic response tends to occur in patients with a previous history of sensitization to minor blood group antigens secondary to pregnancy or a previous blood transfusion. This reaction can result in a severe drop in hemoglobin over 3 to 10 days. The clinical signs and symptoms are not as severe as in hemolytic anemia secondary to ABO incompatibility. As with all hemolytic reactions, the diagnosis is confirmed with positive results on a Coombs test and the identification of alloantibodies in the patient's red blood cells. The management of delayed hemolytic transfusion reactions is nonspecific because the symptoms are usually mild. Symptom control is usually sufficient.

5. Transfusion-Related Acute Lung Injury

Transfusion-related acute lung injury is a clinical syndrome that presents with dyspnea, hypoxemia, cyanosis, hypotension, fever, chills, and noncardiogenic pulmonary edema. This is a severe and sometimes fatal reaction, the third most frequent cause of transfusion-related deaths. The symptoms ordinarily begin 1 to 4 hours after the administration of blood products. The pathophysiology is secondary to the transfusion of HLA-specific antibodies, the passive transfer of which results in increased capillary permeability and microvascular pulmonary damage. Treatment is respiratory supportive care.

6. Transfusion Transmitted Diseases

Nucleic acid screening for human immunodeficiency virus types 1 and 2, hepatitis B and C viruses, and human T-lymphotropic virus types I and II has virtually eliminated these infections from the blood supply, though a statistically calculable risk remains. Bacterial infections still occur in 1 in 2000 platelet transfusions and a smaller proportion of PRBC transfusions. Despite the improvements in screening, the risks have not been completely eradicated. Some of the pathogens transmitted by transfusions include: CMV, *Bartonella* species, *Borrelia* species, *Brucella* species, *Leishmania* species, *Parvovirus*, plasmodia, rickettsiae, *Toxoplasma* species, prions, and certain *Trypanosoma* species.

The common serious culprits of transfusion-related diseases are hepatitis C virus, hepatitis B virus, human immunodeficiency virus, and CMV. Blood donations are routinely screened for both hepatitis viruses and human immunodeficiency virus, but donors who have been recently infected can go undetected. Transfusion recipients may also be at risk for parvovirus B19 and various parasitic infections.

Key Points

Oncologic and Hematologic Emergencies and Complications

- Early admission to the PICU should be considered for children with oncologic complications.

- Preexisting organ dysfunction caused by chemotherapy or its complications is common in pediatric oncology patients.
- Patients with neutropenic fever should be treated aggressively with a single broad-spectrum antimicrobial with antipseudomonal coverage. Colony-stimulating factors should be considered in the patient with septic shock.
- Resistant bacterial or fungal infections are possible in patients in whom fever persists beyond 3 days despite single broad-spectrum coverage.
- High-resolution CT and bronchoalveolar lavage should be considered early in patients with respiratory insufficiency.
- Early institution of a lung-protective strategy, including the use of high-frequency oscillatory ventilation, may improve the outcome in patients with respiratory failure.
- Early evaluation for cardiac dysfunction or adrenal insufficiency is indicated for patients with shock who do not respond to conventional fluid challenges.
- Tumor lysis syndrome – characterized by hyperuricemia, hyperkalemia, hyperphosphatemia, and hypocalcemia – may result in renal failure, seizures, and potentially fatal cardiac arrhythmias.
- Hyperleukocytosis can cause coagulopathy and hyperviscosity, leading to lung injury and stroke. Prompt initiation of cancer therapy is recommended.
- Emergence of leg weakness, paresthesias in the lower extremities, and/or bowel or bladder dysfunction in children with a history of cancer are symptoms suggestive of spinal cord compression.
- Decreased stamina and the presence of headaches, dyspnea, and pallor are signs of anemia.
- The treatment of DIC focuses on the elimination of the catalyst. Replacement therapy consists of the use of platelets, FFP, and cryoprecipitate. Follow-up laboratory studies are necessary to monitor the correction of deficiencies.
- The standard dose of PRBCs is 10 to 15 mL/kg administered over 2 to 4 hours to avoid congestive heart failure. Rapid replacement of PRBCs is indicated in cases of acute blood loss or shock.
- One unit of random platelets per each 10 kg body weight, will increase the platelet count by approximately 50,000/mm^3 provided there is no active consumptive process (fever, immune thrombocytopenic purpura, sepsis, alloimmunization, DIC) or sequestration.
- FFP is administered at 10 to 20 mL/kg and should be infused at a maximum rate of 0.5 mL/kg/min.
- Transfusion-related lung injury presents with dyspnea, hypoxemia, cyanosis, hypotension, fever, chills, and noncardiogenic pulmonary edema. It requires respiratory support.

Suggested Readings

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ACUTE KIDNEY INJURY

Objectives

- Recognize current terminology for stages of acute kidney injury.
- Outline the epidemiology of acute kidney injury.
- Identify risks to renal function associated with systemic illnesses.
- Describe the etiologies of acute kidney injury.
- Determine the use of potential biomarkers.
- Review management of acute kidney injury to prevent mortality.
- Develop a plan to recognize and manage hyperkalemia.
- Differentiate renal replacement therapy modalities.

Case Study

A 4-year-old boy presents with a 5-day history of fever, vomiting, abdominal pain and bloody diarrhea. Though he has maintained oral liquid intake, his urine output has been minimal. His weight on arrival is 17 kg (37.4 lbs), up from 14 kg (30.8 lbs). His vital signs are: blood pressure, 128/65 mm Hg; heart rate, 140 beats/min; respiratory rate, 28 breaths/min; and oxygen saturation on pulse oximetry is 92% on room air. His capillary refill time is 3 to 4 seconds. Examination of the patient is notable for tachypnea with nasal flaring, pallor, irritability, and a diffusely tender, nonrigid abdomen. Initial laboratory results are as follows: hemoglobin, 8.2 g/dL; platelets 90 x 10^9/L; sodium, 143 mEq/L; potassium, 6.2 mEq/L; serum bicarbonate, 13 mEq/L; blood urea nitrogen, 95 mg/dL; and serum creatinine, 1.8 mg/dL.

Detection

– What is this child's physiologic status?

– What is his likely diagnosis?

Intervention

– What are the most immediate treatment strategies?

– What laboratory and imaging studies might be helpful in determining the etiology?

Reassessment

– He receives fluid resuscitation with 20 mL/kg of normal saline in the emergency department. His repeat vital signs are: blood pressure, 125/60 mm Hg; heart rate, 125 beats/min; respiratory rate, 30 breaths/min; and oxygen saturation, 90% on room air. Capillary refill is <2 seconds.

– Is the current treatment strategy effective?

– Does the patient need any other evaluation?

– What next steps are important in preventing further kidney injury?

Effective Communication

– When the patient's clinical status changes, who needs the information and how will it be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How should the treatment strategy be implemented?

– Who is to do what and when?

I. INTRODUCTION

Acute kidney injury (AKI) is a relatively common diagnosis among critically ill children. Previously thought to represent only a sequela of critical illness, AKI is now understood to directly affect both short- and long-term morbidity and mortality. AKI is characterized by a decrease in the renal glomerular filtration rate (GFR), resulting in a decreased ability to eliminate metabolic wastes and regulate electrolyte, acid-base, and fluid homeostasis.

Given the pivotal role AKI plays in critical illness, a high index of suspicion for patients at risk is essential for early identification, determination of causation, mitigation of ongoing injury, and rapid stabilization through goal-directed therapy. This chapter will review the current definitions, epidemiology, pathophysiology, diagnosis, and management strategies for pediatric AKI.

Early identification of patients at risk for AKI is imperative as it may improve clinical outcomes.

II. DEFINITIONS AND CLASSIFICATION

As recognition of the role AKI plays in critical illness has increased, more detailed and specific definitions of AKI have evolved through international consensus efforts. Previously, the pediatric risk, injury, failure, loss, end-stage renal disease (pRIFLE) definition of AKI, which was developed from the adult RIFLE definition, was frequently utilized. At the same time pRIFLE was validated in children, the Acute Kidney Injury Network (AKIN) published a modification of the adult RIFLE definition based on an increase in serum creatinine from a previous trough.

Most recently, the Kidney Disease: Improving Global Outcomes (KDIGO) consortium sought to harmonize prior definitions of AKI and published a comprehensive and inclusive definition (**Table 18-1**). Application of this definition is recommended unless further guidelines are established based on prospective studies. Accordingly, the presence of AKI is determined by the minimum threshold of any of the 3 stage I criteria.

Table 18-1 KDIGO CRITERIA FOR ACUTE KIDNEY INJURY

Stage	SCr-Based	Urine Output
I	Increase ≥0.3 mg/dL (26.4 μmol/L) in 48 hours OR Increase 1.5 – 1.9 times baseline	<0.5 mL/kg/h for 6 – 12 hours
II	Increase 2.0 – 2.9 times baseline	<0.5 mL/kg/h for ≥12 hours
III	Increase ≥3.0 times baseline OR Increase to ≥ 4.0 mg/dL (353 μmol/L) OR If <18 years, decrease in eGFR to <35 mL/min/1.72 m² OR Initiation of renal replacement therapy	<0.3 mL/kg/h for ≥24 hours OR Anuria for ≥12 hours

KDIGO, Kidney Disease: Improving Global Outcomes; SCr, serum creatinine; eGFR, estimated glomerular filtration rate.

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AKI is diagnosed based on the presence of any increase in serum creatinine (SCr), decrease in estimated GFR, or decrease in urine output. In most neonates, AKI is nonoliguric and the SCr measure typically represents maternal creatinine levels, so the definitions apply only to children 1 month of age and older. The KDIGO definitions are beneficial as they provide standardized methods of grading AKI across clinical and research populations. Additionally, several studies have now shown a correlation between these scores and clinical outcomes.

III. EPIDEMIOLOGY

Prevention and treatment of pediatric AKI is aided by an understanding its epidemiology, which may help identify clinical situations that place patients at increased risk for kidney injury. Historically, the use of variable definitions has led to wide-ranging estimates of AKI in hospitalized pediatric patients. However, recent estimates using the KDIGO criteria suggest a prevalence of 12% in children during their ICU course, with approximately 6% of patients with no known kidney disease having AKI at admission. In non-ICU pediatric settings, there is a minimum AKI prevalence of 5%, though standardized measurement is often limited by the absence of serial serum creatinine measurements in otherwise healthy children.

Greater precision in the definition has convincingly revealed that AKI is independently associated with worse outcomes in pediatric patients. Across all stages, both in critically and non-critically ill children, AKI is associated with a longer length of hospital stay. In the ICU, each increase in the stage of AKI is independently associated with increased overall mortality, as well as an increase in duration of mechanical ventilation for those requiring it. Overall 28-day mortality rate increases almost 9-fold for critically ill children with AKI as compared to those without AKI, with a stepwise increase from 18% for stage I to 30% for stage III. The inverse correlation also holds that with clinical improvement and each decrease in AKI stage, overall mortality decreases. Further, an estimated 15% to 20% of patients with AKI require renal replacement therapy (RRT), with the mortality in this subset estimated to be between 30% and 60%. No correlation with overall mortality is noted for children hospitalized in non-ICU settings.

Over the last 2 decades, the incidence of AKI in developed countries has shifted from primary renal diseases (eg, hemolytic uremic syndrome, glomerulonephritis) to a syndrome secondary to other systemic illnesses. In developing countries, however, the most common causes of AKI remain hypovolemia, infection, and primary renal diseases. In high-income countries, this epidemiologic shift has likely been driven by rapid advancements in the medical management of other single and multiorgan system failures. As such, particular groups of patients are known to be at high risk for developing AKI secondary to other systemic illnesses. These groups include patients who: undergo corrective congenital heart surgery, solid organ and stem cell transplant recipients, those with severe illness involving multiorgan dysfunction such as septic shock, and patients exposed to toxins.

IV. RISK FACTORS

In critically ill children, risk factors for AKI can be differentiated by factors present at admission and those present during ICU admission. Admission factors that increase the risk of AKI include: older age, unplanned admission, circulatory or respiratory admission diagnosis, increased illness severity scores, and prior use of nephrotoxic medications. Additional risk factors while in the ICU include: use of new nephrotoxic medications, respiratory dysfunction, and extracorporeal membrane oxygenation treatment. Common nephrotoxic medications include: antibiotics, antimycotics, chemotherapeutics, nonsteroidal anti-inflammatory drugs (NSAIDs), and angiotensin-converting enzyme (ACE) inhibitors. As these medications are prescribed by healthcare providers, they represent a significant category of modifiable risk and preventable harm.

One of the most frequent causes of – and risk factors for – AKI is sepsis. Sepsis-associated AKI is a

syndrome in which clinical criteria are met for both sepsis and AKI. The incidence of sepsis-associated AKI is estimated to be between 9% and 34%, representing a significant portion of pediatric AKI cases. The mechanism of injury is complex and likely multifactorial in this setting, with blood flow alterations due to intravascular hypovolemia and renal hypoperfusion as well as secondary to pro-inflammatory cytokines. In patients with severe sepsis, those with AKI have a 2-fold increase in mortality compared to those without AKI, further underscoring the significant role AKI plays in critical illness.

Baseline risks, acute clinical conditions, and diagnostic/therapeutic agents associated with the development of in-hospital AKI are shown in **Table 18-2**.

Table 18-2	RISK FACTORS FOR ACUTE KIDNEY INJURY	
Baseline Risks	**Acute Clinical Conditions**	**Nephrotoxic Agents**
Chronic kidney disease	Abdominal compartment syndrome	Antimicrobial agents
Congenital heart disease	Hypotension/shock	Chemotherapeutic drugs
Heart failure	Hypovolemia	Contrast media
Hypoalbuminemia	Mechanical ventilation	Immunosuppressive agents
Liver failure	Nonrenal solid organ transplant	Nonsteroidal anti-inflammatory drugs
	Rhabdomyolysis	
	Sepsis	
	Stem cell transplant	

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V. PATHOPHYSIOLOGY

Despite the shifting definitions and epidemiology of AKI, pathologic understanding and diagnostic evaluation are facilitated by classifying AKI into three broad categories: prerenal, intrinsic renal, and postrenal or obstructive AKI. Together, prerenal and intrinsic renal causes account for ~90% of all AKI. These categories can assist healthcare providers in narrowing the differential diagnosis and allowing for more targeted therapeutic intervention. **Table 18-3** lists common causes of AKI according to this classification.

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Differentiation of prerenal, intrinsic renal, and postrenal AKI is important to determining the underlying etiology.

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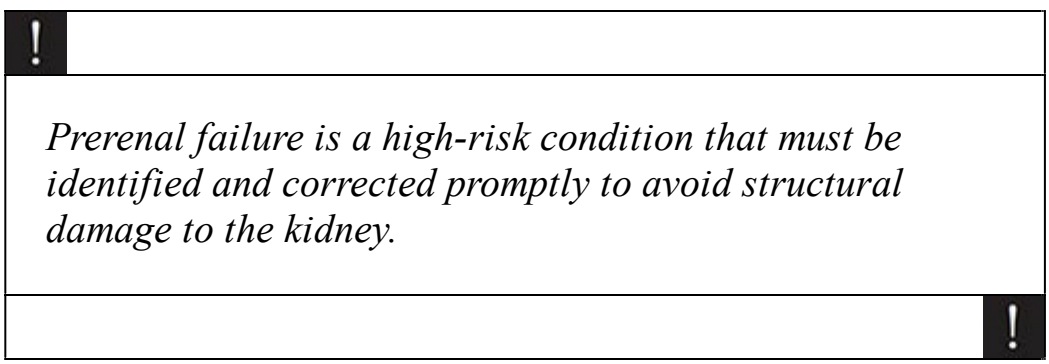
Table 18-3	COMMON ACUTE KIDNEY INJURY ETIOLOGIES
Prerenal	• Intravascular volume depletion - Reduction in blood volume: hemorrhage, increased GI/renal losses, burns - Redistribution of blood volume: nephrotic syndrome, liver failure, sepsis, malnutrition • Loss of vascular tone: anaphylaxis, shock • Decreased effective blood delivery: heart failure, congenital heart disease, low cardiac output • Increased resistance to flow: renal artery stenosis, abdominal compartment syndrome
Intrinsic	• Tubular - Ischemia/progressive prerenal AKI: sepsis or septic shock - Endogenous nephrotoxins: rhabdomyolysis, hemolysis, tumor lysis syndrome • Interstitial: drug-induced, allergic, or idiopathic • Glomerular: post-infectious, immune-mediated (eg, systemic lupus erythematosus) • Vascular: hemolytic uremia syndrome, cortical bilateral necrosis, renal or arterial thrombosis
Post-renal	• Ureteral obstruction: nephrolithiasis, ureteropelvic junction obstruction, tumor, or mass • Bladder obstruction: neurogenic bladder, tumor • Urethral obstruction: posterior urethral valves, urinary catheter obstruction • Congenital anomalies

Abbreviations: GI, gastrointestinal; AKI, acute kidney injury

A. Prerenal AKI

In the healthy state, the kidneys receive 20% to 25% of systemic cardiac output. Thus, factors that decrease the flow of blood – volume and/or rate – to the kidneys predispose to prerenal AKI. Prerenal causes account for ~20% of all AKI. Under conditions of hypoperfusion, the healthy kidney responds by increasing tubular reabsorption of both sodium and water through multiple mechanisms.


!
Prerenal failure is a high-risk condition that must be identified and corrected promptly to avoid structural damage to the kidney.
!

Hypovolemia, or a decrease in effective arterial volume, restricts renal perfusion and adversely affects renal function. Causes include inadequate intake, hemorrhage, intestinal obstruction, gastrointestinal fluid losses, severe burns, decompensated nephrotic syndrome, and trauma. Hypovolemia can also arise as a result of polyuria, which may be caused by osmotic diuresis in diabetic ketoacidosis, the different forms of diabetes insipidus, a salt-wasting syndrome, or mineralocorticoid deficiency.

A fall in effective arterial volume is also found in heart failure and any situation involving a

significant decrease in cardiac output. For many pediatric surgical patients, risk factors also include the duration of the surgery and the amount of blood lost. Specifically, in the postoperative course of cardiovascular surgery, the underlying cardiac pathology, procedure, and duration of cardiopulmonary bypass or aortic cross-clamp are all factors that may increase the risk for AKI. In patients with low effective arterial volume in whom filtration is maintained at the expense of vasoconstriction of the efferent arteriole, vasodilators – like angiotensin-receptor blockers or ACE inhibitors – may cause a sharp drop in filtration.

B. Intrinsic Renal AKI

Intrinsic renal AKI is further categorized based on the 4 primary renal elements involved: tubules, interstitium, glomeruli, and intrarenal blood vasculature.

Acute tubular necrosis (ATN) describes injury to the renal tubules and accounts for 40% to 50% of the causes of AKI. The most common cause of ATN is progression of prerenal AKI through sustained hypoperfusion. Thus, mechanisms that decrease renal blood flow and predispose to prerenal AKI also may result in ATN if not recognized and reversed rapidly. Overall, ischemic or nephrotoxin-related renal injury accounts for ~80% AKI cases and often occurs through multiple pathophysiologic mechanisms simultaneously.

Interstitial injury is known as *acute interstitial nephritis* and is primarily caused by toxins or nephrotoxic agents. The risk of acute interstitial nephritis associated with contrast media persists, particularly in patients with preexisting dehydration or renal compromise, and in those who receive a relatively large dose of contrast. Other drugs most commonly associated with nephrotoxicity include NSAIDs, ACE inhibitors, angiotensin-receptor blockers, aminoglycosides, vancomycin, amphotericin B, IV immunoglobulin, acyclovir, and immunosuppressive calcineurin inhibitors. Rhabdomyolysis can generate AKI, though it has not been clearly established at what levels of creatine kinase and myoglobin this occurs. Myoglobin in the presence of hypovolemia or ischemia is particularly nephrotoxic. Similarly, tumor lysis syndrome can also precipitate AKI, especially in children with lymphoma, who may even develop it before beginning therapy because of cellular autolysis.

Glomerulonephritis is often the result of a primary immune complex-mediated disease, such as post-infectious glomerulonephritis or a systemic autoimmune inflammatory condition, such as systemic lupus erythematosus. In these acute glomuleronephritides, immune complexes are deposited within the glomerulus either in response to external (eg, infectious) or one's own antigens. Subsequent activation of the complement system, leukocyte recruitment, and cytokine release lead to glomerular inflammation and injury.

Vascular causes of intrinsic AKI include malignant hypertension, atherothrombotic disease, and hemolytic uremic syndrome (HUS)/thrombotic thrombocytopenic purpura. HUS is a relatively common cause of intrinsic AKI in children. It typically presents as microangiopathic hemolytic anemia and thrombocytopenia associated with oliguria or anuria, often preceded by bloody diarrhea. The cause is frequently an intestinal infection by a verotoxin-producing strain of *Escherichia coli*. HUS may have a multisystemic course that involves the central nervous system, colonic wall, pancreas, or myocardium.

C. Postrenal AKI

Urinary tract obstruction can cause kidney damage and loss of function. Bilateral ureteral obstruction, obstruction of a solitary kidney, or urethral obstruction by valves, a clot, or ureterocele may be responsible.

VI. DIAGNOSIS

A. Clinical Diagnosis

An accurate history provides data on underlying chronic illness, as well as the temporal relationship between acute inciting events and the onset of AKI. The physical exam can give clues regarding underlying systemic diseases involving the kidney. Hypertension may suggest underlying chronic kidney disease. Tachycardia and hypotension suggest decreased circulatory volume and likely renal hypoperfusion. The presence of peripheral edema and hypervolemia (heart failure) suggests decreased cardiac output and is a risk factor for renal injury. Cutaneous signs, such as petechiae or purpura, may suggest a diagnosis of HUS or vasculitis.

Although urinary output changes can be present in most types of AKI, this readily available information is valuable because the treatment of nonoliguric renal failure is quite different from that of oligoanuric failure. Anuria is indicative of a complete obstructive lesion, vascular lesion, rapidly progressive glomerulonephritis, or bilateral cortical necrosis. When seen with bladder distention, it is suggestive of an obstructive uropathy, although obstruction can cause AKI without causing anuria.

B. Laboratory Diagnosis

Initial laboratory studies should include the following: basic electrolyte panel, including phosphorus, magnesium, and ionized calcium; blood urea nitrogen (BUN); SCr; and complete blood cell count. Urine studies should include urinalysis, microscopy, urine sodium, urine protein and creatinine, and osmolality. Many of these laboratory values can be utilized to calculate other indices for the classification of AKI; these indices are straightforward to calculate and are readily available at the bedside.

Table 18-4 CLINICAL SITUATIONS THAT INFLUENCE BLOOD UREA NITROGEN AND CREATININE VALUES

	Factors That Increase	Factors That Decrease
Blood urea nitrogen	Hypercatabolic states Sepsis Starvation Gastrointestinal bleeding High protein diet Dehydration Renal hypoperfusion	Malnutrition Low protein diet Liver failure Nephrotic syndrome Hypometabolism
Creatinine	Rhabdomyolysis	Liver failure

	Cimetidine Tumor lysis syndrome Dehydration	Loss of muscle mass Hyperlipidemia

The upper normal creatinine values vary with age. Both creatinine and BUN values can be higher or lower depending on the specific clinical conditions (**Table 18-4**). Normal values for both are shown in **Appendix 1**. Other metabolic alterations seen in patients with AKI include:

- Metabolic acidosis with wide anion gap due to retention of sulfates, phosphates, and other anions
- Hyperkalemia
- Hyponatremia
- Hyperphosphatemia and hypocalcemia
- Hypermagnesemia

Table 18-5 URINARY INDICES IN ACUTE KIDNEY INJURY

AKI Classification	Urine Volume	BUN/Cr Ratio	Urine-specific Gravity	FENa	FEurea
Prerenal AKI	≤0.5 mL/kg/h	>20	>1.020	<1%	<35%
Intrinsic AKI	Variable	Variable	1.010-1.020	>3%	>35%

Abbreviations: AKI, acute kidney injury; BUN, blood urea nitrogen; Cr, creatinine; FENa, fractional excretion of sodium; FEurea, fractional excretion of urea.

One index that points to an appropriate renal response mechanism, and therefore intact tubular function, is an increase in the BUN/creatinine ratio, typically greater than 20:1 in states of reduced renal perfusion. A similar index is the fractional excretion of sodium (FENa). When intrinsic renal function is intact, sodium reabsorption by the renal tubule increases, the urine sodium falls to <20 mEq/L, and the FENa is <1%, indicating the presence of prerenal AKI. In contrast, in ATN or intrinsic forms of AKI, the FENa is typically >3% (**Table 18-5**). The calculation of the FENa requires simultaneous measurements of the serum sodium (SNa) and SCr, urine sodium (UNa), and urine creatinine (UCr) for use in the formula:

$$\text{FENa (\%)} = (\text{UNa} \times \text{SCr}) / (\text{SNa} \times \text{UCr}) \times 100$$

Extrinsic influences, such diuretic therapy, can also disrupt this normal physiologic response, affecting urinary sodium concentrations and decreasing the accuracy of the FENa as a marker for prerenal AKI. In such cases, the fractional excretion of urea (FEurea) can be used. Calculation of FEurea is the same as that for the FENa with the substitution of plasma (PUrea) and urine urea (Uurea) for plasma and urine Na:

$$\text{FEurea (\%)} = (\text{Uurea} \times \text{SCr}) / (\text{PUrea} \times \text{UCr}) \times 100$$

In prerenal AKI, the FEurea is typically <35%.

Urinalysis and microscopic evaluation of the urinary sediment can provide clues to the underlying

etiology of AKI. Urinalysis in prerenal azotemia is often normal except for occasional fine granular and hyaline casts. Microscopic evaluation in intrinsic tubular injury, or ATN, shows epithelial cell casts and coarse granular casts. The presence of white blood cells and white blood cell casts is indicative of active inflammatory processes, such as glomerulonephritis, infection, or acute tubulointerstitial nephritis. Urine eosinophils are classically associated with the latter condition but can be seen in many other causes of AKI, such as glomerulonephritis and cystitis. Albuminuria and red blood cell casts indicate underlying glomerular disease.

It is important to note that elevation in SCr represents a relatively late biomarker finding of kidney injury as an increase may not be noted until >50% of renal function has been compromised. Additionally, SCr may fluctuate based on patient factors unrelated to kidney injury, such as diet, gender, age, muscle metabolism, and hydration status, among other factors. Given this potential delay in clinical diagnosis, new biomarkers – such as neutrophil gelatinase-associated lipocalin, kidney injury molecule 1, interleukin 18, and liver fatty acid-binding protein, among others – are being evaluated for use in the early diagnosis of AKI. **Table 18-6** outlines some of the biomarkers under evaluation. While some of these may be clinically available at certain sites, none has been widely adapted to the definitions of AKI. Future revisions to the KDIGO criteria are likely to incorporate biomarkers that are shown to provide superior sensitivity and specificity to creatinine in a more timely fashion.

Table 18-6 BIOMARKERS IN ACUTE KIDNEY INJURY

Biomarker Type	Biomarker
Functional marker	Serum creatinine Serum cystatin C Urine albumin
Up-regulated proteins	Neutrophil gelatinase-associated lipocalin (NGAL) Kidney injury molecule 1 (KIM-1) Liver-type fatty acid–binding protein (L-FABP) Interleukin 18 (IL-18) β-trace protein (BTP) Asymmetric dimethylarginine (ADMA)
Low-molecular-weight proteins	Urine cystatin C
Enzymes	N-acetyl-glucosaminidase (NAG) Glutathione-S-transferase (GST) Gamma-glutamyl transpeptidase (GGT) Alanine aminopeptidase (AAP Lactate dehydrogenase (LDH)

C. Diagnostic Imaging

Radiologic imaging of the kidney is also important in the evaluation of a patient with AKI. Renal and bladder ultrasound, the most commonly used initial study, provides information on renal length and size, as well as signs of obstruction (ie, hydronephrosis, ureteral and bladder dilatation) and changes in bladder wall contour. Small atretic kidneys can suggest underlying chronic kidney disease. Increased echogenicity is an often-noted finding in AKI, although it is a nonspecific characteristic. A Doppler ultrasound may be helpful to rule out renal vascular lesions such as renal artery stenosis, but

if the index of suspicion remains high in the face of a negative study, additional imaging modalities may be indicated. Detection and monitoring of hydronephrosis is one of the most common utilizations of serial ultrasound. In the face of new AKI, bilateral hydronephrosis should raise suspicion for distal obstruction that requires rapid identification and intervention.

VII. PREVENTION AND TREATMENT

At present, no specific medical therapies have been shown to treat AKI. Prevention and prompt recognition are paramount to limiting AKI in its early stages. ICU management includes careful attention to volume status, restoration and maintenance of renal perfusion, correction of acidosis and electrolyte abnormalities, and nutritional support.

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The objectives of AKI management in children are to maintain fluid, electrolyte, and metabolic homeostasis; prevent progression of the renal disease; treat the underlying cause of AKI; and provide adequate nutritional support.

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A. Fluid Volume Management

Initial evaluation should include assessment of the intravascular volume status. Many patients with AKI present in a hypovolemic state, and volume expansion to achieve euvolemia with isotonic crystalloids is the mainstay of initial care. Following volume expansion, strict monitoring of all inputs and outputs is paramount. Indwelling urinary catheters are not necessary in many patients and should be avoided to reduce the risk of infection. Urinary catheters may still be indicated in certain subsets of patients, including young children with gastroenteritis (to differentiate urinary from stool losses) and suspected oliguria or anuria; catheters should be removed soon as possible to avoid catheter-associated urinary infections. Weight should be followed daily as another indicator of fluid status.

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Maintenance of adequate renal perfusion is one component of the management strategy.

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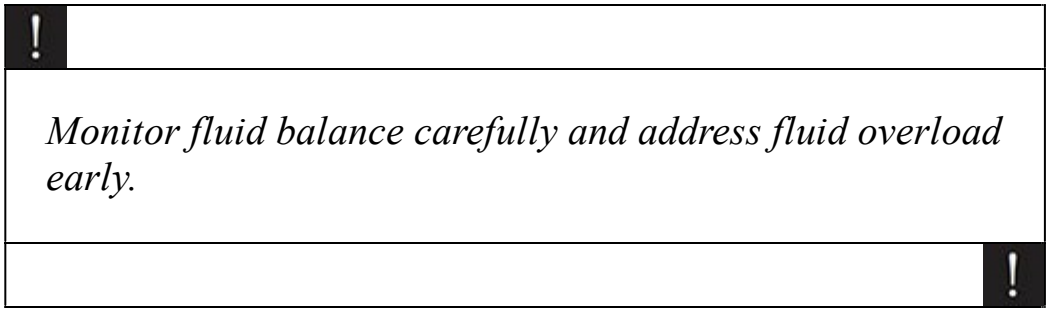
In addition to fluid administration, inotropic and vasoactive medications may be required to maintain

renal blood flow. Infusions should be titrated to target age-appropriate normal blood pressures.

Once volume resuscitation has been achieved, ongoing fluid management includes maintenance of euvolemia. Recent data suggest fluid overload in patients with AKI is related to poorer outcomes, including death, and thus should be avoided when possible. Ongoing fluid needs should be calculated to provide insensible losses (ie, 400 mL/m²/day) in addition to the replacement of urine output and other measurable losses, such as nasogastric output.



Monitor fluid balance carefully and address fluid overload early.

One method for assessing the degree of fluid overload is to monitor fluid balance from admission in relation to the child's estimated dry weight using the formula:

$$\% \text{ Fluid overload} = \frac{[\text{Fluid intake in liters}] - [\text{Fluid output in liters}]}{\text{Admission weight in kg}} \times 100\%$$

Studies have shown that patients with >20% overload are at increased risk for death, and additional therapies − including diuresis and RRT − should be considered. Diuretics have not been shown to have a definite benefit in AKI, despite their widespread administration. They may be used judiciously to minimize fluid overload. However, as diuretics can have direct and indirect nephrotoxic effects, they must be used with caution while carefully monitoring renal function.

Vasoactive therapies, including low-dose dopamine and fenoldopam, have been proposed to augment renal perfusion by vasodilation of the renal vasculature. However, studies to date do not clearly support this approach, and it should not be used in the typical patient with AKI.

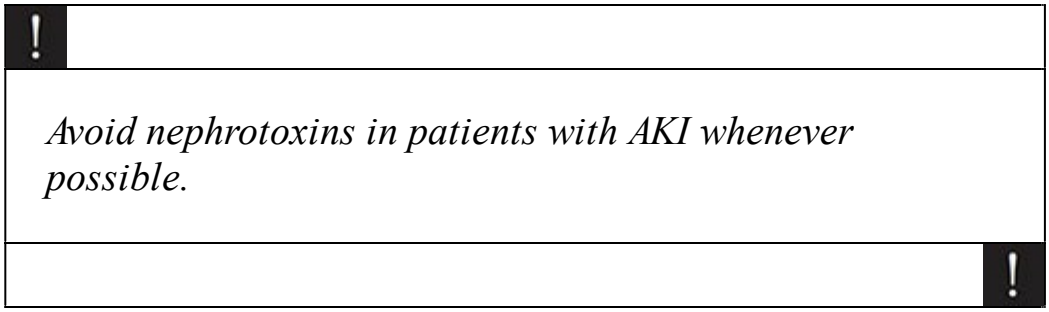
B. Avoidance of Nephrotoxicity

The list of pharmacologic agents that are potentially injurious to the kidney is extensive. Some of the most commonly utilized nephrotoxins are aminoglycoside antibiotics, antifungals, NSAIDs, chemotherapeutics, and radiocontrast dyes. Avoidance of these agents whenever possible is one element of preventing AKI. When use is unavoidable, dosage adjustments should be made for the level of kidney function. Therapeutic levels should be followed for medications with readily available serum levels. An estimate of GFR can be calculated with the Schwartz formula, as follows:

$$\text{GFR} = \frac{k \times \text{Height (cm)}}{\text{SCr}}$$

with k values determined by age: infant = 0.45; 1-13 years and adolescent girls = 0.55; adolescent boys = 0.7.

This formula is only an estimate of GFR and tends to overestimate true renal function in many clinical situations. An accurate estimate of GFR can only be obtained when SCr is at a steady state. Therefore, with a rising SCr, doses should be adjusted to the value for the lowest range of kidney function for that agent.



C. Electrolyte Management

As noted previously, a hallmark of AKI is the inability to maintain electrolyte homeostasis. Common metabolic derangements include hyperkalemia, metabolic acidosis, hyperphosphatemia, hypocalcemia, and hypermagnesemia. Therapeutic interventions for these derangements in patients with AKI are discussed below. Please also refer to **Chapter 8** for a more in-depth discussion. Patients with electrolyte abnormalities may be asymptomatic or present with only subtle symptoms. Therefore, close monitoring of serum levels is important during AKI.

1. Hyperkalemia

Hyperkalemia is a common derangement in AKI, and the most concerning, as it can lead to life-threatening arrhythmias. The hyperkalemia in AKI is secondary to reduced renal filtration and reduced potassium excretion. Additional factors resulting in elevated serum potassium levels include extracellular shifting of potassium due to metabolic acidosis, and release from cells due to tumor lysis or rhabdomyolysis.

First-line therapy in a patient with AKI is prevention and management of hyperkalemia. Potassium administration should be discontinued in both intravenous and enteral forms, and frequent monitoring of potassium levels should be performed. Continuous electrocardiographic (ECG) monitoring may be useful in early identification of a rising potassium level.

a. Mild to Moderate Hyperkalemia

Potassium levels between 5.1 and 6.5 mEq/L typically present without associated ECG changes. Patients with adequate urine output may often be managed by discontinuation of potassium intake and IV fluid administration. Patients at the higher end of the range will benefit from medications that remove potassium from the body. These include loop diuretics, which work by augmenting urine output and potassium secretion, as well as sodium polystyrene sulfate buffers given via the gastrointestinal tract, which eliminate potassium via an exchange mechanism with sodium. Serial monitoring of serum potassium and continuous ECG monitoring are obligatory.

b. Severe Hyperkalemia

At potassium levels over 6.5 mEq/L, ECG changes may occur and patients are at risk of sudden life-threatening decompensation. Typical changes include peaking of T waves and prolongation of the PR interval, followed by flattening of the P waves and widened QRS complexes. This rhythm degenerates into a sine wave pattern and ventricular tachycardia if not rapidly corrected. As these changes result from alteration of the transmembrane resting potential in cardiac myocytes, therapy is directed at restoration of the potential. This is achieved in a multifactorial manner, by increasing serum calcium levels and reducing serum potassium levels.

Calcium administration is the most rapid method for restoring membrane potential. Calcium gluconate is preferred when administered peripherally; calcium chloride may be given in patients with central venous access.

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In a patient with hyperkalemic ECG changes, careful calcium infusion is the quickest way to restore cardiac stability.

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Methods to rapidly shift potassium out of the serum and into the intracellular space include administration of sodium bicarbonate, insulin, and β-agonists.

- Calcium gluconate: 100 mg/kg dose IV over 5 minutes
- Calcium chloride: 20 mg/kg dose IV push via central line
- Sodium bicarbonate: 1 to 2 mEq/kg IV over 5 to 10 minutes
- Insulin: 0.1 U/kg/h concurrently with dextrose 25%, 2 mL/kg/h, to prevent hypoglycemia
- Sodium polystyrene sulfonate: 1g/kg rectally or orally
- β-agonist nebulized solution (ie, albuterol)

As in patients with mild to moderate hyperkalemia, action should also be taken to remove potassium from the body. Loop diuretics and sodium polystyrene sulfate are the most commonly used agents. Patients who are receiving dialysis should be dialyzed quickly, and renal replacement therapy should be considered all patients with life-threatening hyperkalemia. Different approaches in the ICU include peritoneal dialysis and continuous venovenous hemofiltration.

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Digitalis intoxication can cause severe hyperkalemia and should be treated with a digoxin-binding immune product - NOT with calcium infusion. Digitalis toxicity can be exacerbated by calcium infusion.

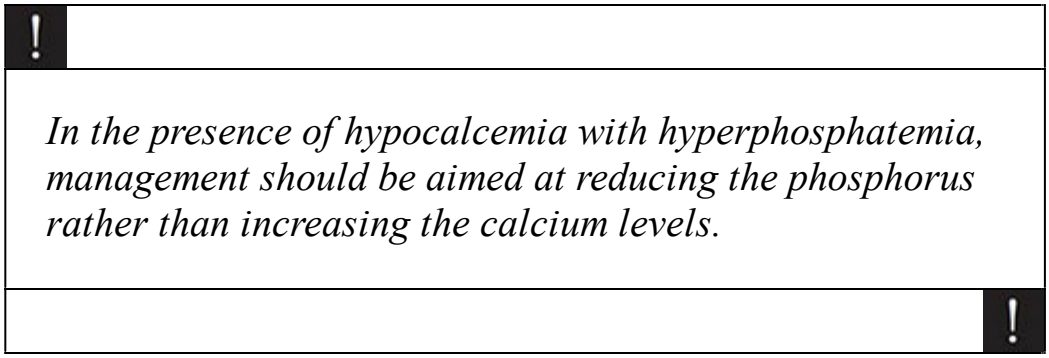
The healthcare provider should be aware that the serum potassium level causing life-threatening cardiac changes will vary among individuals.

2. Metabolic Acidosis

Metabolic acidosis from AKI is secondary to an inadequate excretion of hydrogen ions, causing the consumption of bicarbonate as a buffer. The degree of acidosis may be impacted by other ongoing causes of acidosis in the critically ill patient, including hypoperfusion. Severe acidosis can increase myocardial irritability and affect peripheral vascular resistance, enzyme activity, and energy production. While most therapy for acidosis in the ICU is aimed at resolving the underlying cause, bicarbonate replacement may be useful in AKI. Mild to moderate metabolic acidosis with a pH > 7.20 can be treated with oral bicarbonate supplements. Severe metabolic acidosis with pH < 7.20 or metabolic acidosis in the presence of hyperkalemia should be corrected intravenously. Correcting a low pH in renal failure risks decreasing ionized calcium, a critical problem, particularly if the calcium is already low due to phosphate retention. If given, IV sodium bicarbonate should be administered slowly (in doses of 1 mEq/kg) while monitoring sodium and ionized calcium.

3. Hyperphosphatemia

Hyperphosphatemia is the result of reduced excretion during AKI. The excess circulating phosphorus can bind to free calcium, reducing serum calcium levels. Dietary phosphorus intake should be limited, and oral phosphate binders may be given to reduce the amount of phosphorus absorbed in the gastrointestinal tract.


In the presence of hypocalcemia with hyperphosphatemia, management should be aimed at reducing the phosphorus rather than increasing the calcium levels.

4. Hypocalcemia

Hypocalcemia, the result of free calcium binding phosphorus, is exacerbated by the kidney's failure to convert inactive to active vitamin D. Monitoring free calcium is important, as this is the metabolically active component of the serum calcium. Typically during AKI, calcium is replaced only in symptomatic patients or those with extremely low free calcium levels. For routine replacement, calcium gluconate, 100 mg/kg IV, is the preferred therapy.

5. Hyponatremia

Inadequate glomerular filtration may result in fluid retention and hyponatremia. Patients with severe hyponatremia or those with symptoms may require correction of the sodium deficit. In most cases, careful fluid management is sufficient to correct sodium abnormalities.

$$\text{Sodium deficit} = 0.6 \times \text{weight (kg)} \times (\text{target sodium} - \text{measured sodium})$$

D. Renal Replacement Therapy

When the fluid and electrolyte abnormalities cannot be corrected with medical management, renal replacement therapy may be indicated (**Table 18-7**).

Table 18-7 ADDITIONAL INDICATIONS FOR RENAL REPLACEMENT THERAPIES

Fluid Overload	Electrolyte or Acid-Base Disorders	Toxin Removal
• Pulmonary edema • Congestive heart failure • Persistent anuria or oliguria • Refractory hypertension • Oliguria and high fluid requirements • Oliguria requiring negative fluid balance • >20 % fluid overload	• Severe hypo- or hypernatremia • Hyperkalemia (K^+ ≥7 mEq/L) • Persistent, severe acidosis	▪ Urea ▪ Uric acid ▪ Exogenous dialyzable toxins

Peritoneal dialysis is a modality in which a catheter is inserted percutaneously or, preferably, surgically into the abdominal cavity, utilizing the peritoneal membrane as a dialysis filter. Solute clearance occurs via diffusion due to concentration gradients between the vascular space and the infused dialysis solution. Similarly, fluid removal is driven by an osmotic gradient created by dextrose contained in the infused dialysate. Peritoneal dialysis does not require central venous access or anticoagulation and can be performed continuously if needed for slower correction of electrolyte derangements and slower fluid removal. It is also a good option in patients too small for venous dialysis catheters. Peritoneal dialysis can be limited by prior abdominal surgeries and congenital abdominal wall anomalies and is less well-suited for removal of ingested toxins. Peritoneal dialysis remains the predominant acute dialytic modality in developing countries. It should be considered as the first option in special cases: neonates, patients with difficult vascular access, patients with hemodynamic instability, and children who have undergone cardiac surgery.

The general term *hemodialysis* describes a modality in which an external pump moves a patient's blood through an extracorporeal circuit and across an artificial membrane, allowing for both fluid removal and correction of metabolic derangements. Hemodialysis can be performed either as intermittent hemodialysis (IHD) or continuous renal replacement therapy (CRRT).

In IHD, the patient receives therapy during a prescribed period of hours and then is off therapy until the next session. The finite period requires that solute and fluid removal occur fairly rapidly; therefore, it is more suited to the hemodynamically stable patient who can tolerate rapid changes in volume status. Some benefits of IHD include its efficiency in correcting inborn metabolic abnormalities and toxic ingestions. It also allows patient mobilization for necessary diagnostic testing

and conditioning, such as physical therapy.

For those patients with hemodynamic instability or large volumes of fluid overload, fluid removal and solute correction at a slower rate is more appropriate, and CRRT may be a better approach. CRRT takes place 24 hours each day, so that correction of the fluid balance is slower and can be adjusted throughout the treatment in response to changes in the patient's clinical status (such as hypotension). CRRT requires anticoagulation to the circuit, which may be performed with heparin or citrate. Although no direct comparison of IHD and CRRT has shown CRRT to be superior in clinical outcomes, its utility in hemodynamically unstable patients typically makes it the first-line therapy in the ICU.

Achieving good vascular access is a key step in the provision of either IHD or CRRT. The preferred site for central venous catheter placement is the internal jugular vein, followed by the femoral vein. The largest lumen catheter that can be safely placed will allow attainment of prescribed blood flows and decrease the risk of clotting. Maintaining vascular access in patients requiring dialysis is a matter of life-saving importance.

The pharmacokinetics of drug clearance on dialysis can be variable, requiring vigilant monitoring of drug levels and adjustment of dosing based on clinical response. Consultation with the ICU pharmacist can be extremely helpful in managing dosing adjustments.

E. Nutritional Support

Children with AKI have an increased rate of catabolism and are at risk of malnutrition. Adequate nutrition is essential for the treatment of AKI, but providing sufficient nutrition may be difficult because of fluid restrictions.

Concentrated enteral or parenteral nutrition should be used. The enteral route is always preferred when available. Available enteral formulas provide lower phosphorus and potassium loads, limiting electrolyte abnormalities. When necessary, total parenteral nutrition may be given with careful attention to minimizing the fluid load as much as possible. Lipids and concentrated glucose solutions improve caloric concentration. Essential amino acids can be used to decrease urea production. Initial protein intake should be limited to 0.5 to 1 g/kg/day. In some situations, dialysis may be utilized to remove the extra fluid required to provide sufficient caloric intake.

F. Management of Hypertension

Hypertension is often secondary to volume overload in patients with oliguria. Dialysis is indicated in hypervolemic, hypertensive patients who fail to respond to antihypertensives, fluid restriction, and diuretics. Those with severe hypertension and encephalopathy or cardiomyopathy should be treated aggressively (**Table 18-8**). Less severe conditions can be treated with intermittent therapy. Doses of antihypertensive agents may need to be adjusted to the GFR.

Table 18-8	USEFUL MEDICATIONS IN HYPERTENSIVE EMERGENCIES

Medication	Loading Dose	Route	Frequency	Starting Maintenance	Maximum Dose
Esmolol	100–500 µg/kg over 1 minute	IV	Continuous	100 µg/kg/min	500 µg/kg/min
Hydralazine	0.15–0.2 mg/kg	IV, IM	Every 4–6 hours	Not applicable	20 mg/dose
Labetalol[1]	0.2–1 mg/kg over 2 minutes	IV	Every 10 minutes	0.25–1 mg/kg/h	20 mg/dose; 3 mg/kg/h
Nicardipine[2]	None	IV	Continuous	0.5–1 µg/kg/min	5 µg/kg/min
Nitroprusside[3]	None	IV	Continuous	0.3–0.5 µg/kg/min	10 µg/kg/min

[1] Labetalol is relatively contraindicated with asthma and acute heart failure.

[2] Nicardipine may cause reflex tachycardia.

[3] Thiocyanate levels should be monitored during prolonged (>72 hour) sodium nitroprusside usage.

VIII. LONG-TERM OUTCOMES

While pediatric data are limited, children surviving AKI may have an increased risk of long-term changes in kidney function, microalbuminuria, and hypertension. In a study following patients 3 to 5 years after AKI, nearly 60% persisted in some degree of underlying renal dysfunction. A significant comorbidity likely is associated with surviving AKI, emphasizing the need for new methods of early diagnosis, prevention, and treatment. The complexity of managing AKI in the face of critical illness requires early consideration to providing nephrology expertise and resources. Transport (**Chapter 14**) to a pediatric center may be necessary to give these children the best chance of quality survival.

Key Points

Acute Kidney Injury

- AKI occurs commonly in critically ill children and significantly increases morbidity and mortality rates.
- Sepsis and nephrotoxic medications are common causes of AKI.
- Traditional indicators used to differentiate prerenal from renal failure may support the diagnosis of AKI during the initial phase, before the patient has received diuretics or vasoactive drugs. They often are not useful in the critically ill child who needs multiple pharmacologic therapies.
- Metabolic acidosis, hyperkalemia, and hypocalcemia are metabolic conditions frequently found in patients with AKI.
- Strategies to avoid primary or secondary renal injury consist mainly of ensuring adequate renal blood flow and avoiding nephrotoxicity.
- Patients should be monitored for signs of fluid overload. Strict fluid balance and daily weight

records are essential.

- Broad indications for renal replacement therapies include fluid overload, electrolyte or acid-base disorders, and toxin removal unresponsive to medical therapies.
- Choice of renal replacement therapy should be based on overall clinical status and the goals of the therapy.
- Diligence in drug dosing is required in AKI and dialysis.
- An ideal nutrition plan for AKI patients would provide adequate caloric intake with fluid restriction, limit protein intake, and reduce or eliminate potassium intake.
- AKI can have long-term sequelae.

Suggested Readings

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POSTOPERATIVE MANAGEMENT

Objectives

- Discuss the effect of anesthesia on patients during the postoperative period.
- Recognize and manage common postoperative complications.
- Describe a multimodal approach to postoperative pain management.
- Summarize the fluid, electrolyte, and nutritional management.
- Review management of postoperative nausea and vomiting.
- Discuss thermal regulation and evaluation of postoperative fever.

Case Study

A 2-year-old boy is admitted because of intermittent crying episodes, abdominal distension, and bilious emesis. He undergoes exploratory laparotomy with surgical findings consistent with malrotation. His past medical history is significant for asthma, which is stable with the use of albuterol and a budesonide/formoterol inhaler. Within 30 minutes of arrival to the floor, he had 2 episodes of nonbloody, nonbilious vomiting. His vital signs were: temperature, 38.3°C (100.9°F); respiratory rate, 10 breaths/min; heart rate, 130 beats/min; blood pressure, 74/50 mm Hg; and pulse oximetry saturation, 89% on room air. You are called to the bedside to evaluate the patient.

Detection

– What would you want to know about his preoperative and intraoperative course?

– What is the most likely etiology of the bradypnea?

– What are the possible causes for the desaturation?

Intervention

– What is the treatment strategy for postoperative bradypnea?

– What would be the best approach to pain management?

Reassessment

– How would you assess the effectiveness of the patient's respirations?

– What are the potential respiratory complications you might encounter?

Effective Communication

– What should be communicated in the handoff between the operating room team and the ward team assuming care for the patient?

Teamwork

– Who else might need to be involved in the care of the patient?

I. INTRODUCTION

The patient's preoperative clinical condition and management, along with the intraoperative course, determine the postoperative physiologic status and disposition. Key concepts include fluid and electrolyte therapy, analgesia, and cardiopulmonary evaluation and management. This chapter attempts to integrate these concepts and relate them specifically to the postoperative management of the pediatric patient.

II. PREOPERATIVE AND ANESTHETIC CONSIDERATIONS

With the advent of modern anesthetic techniques and specialty training in pediatric anesthesiology, anesthetic-related deaths in children are uncommon and estimated at 1 in 185,000 patients. The risk of anesthesia-related cardiac arrest increases in infants who are younger than 1 year and in patients with severe underlying disease. Most deaths occur in sick children undergoing emergency surgery. The preoperative severity of illness remains a significant risk factor. The American Society of Anesthesiologists (ASA) developed the physical status classification to estimate the severity of illness prior to administration of sedation or anesthesia (**Chapter 20**). This classification does not predict risk, but allows the anesthesiologist to tailor the anesthetic to the patient's underlying condition. Therefore, the higher the ASA class, the greater the likelihood that the patient will have a complicated postoperative course and require intensive management and monitoring.

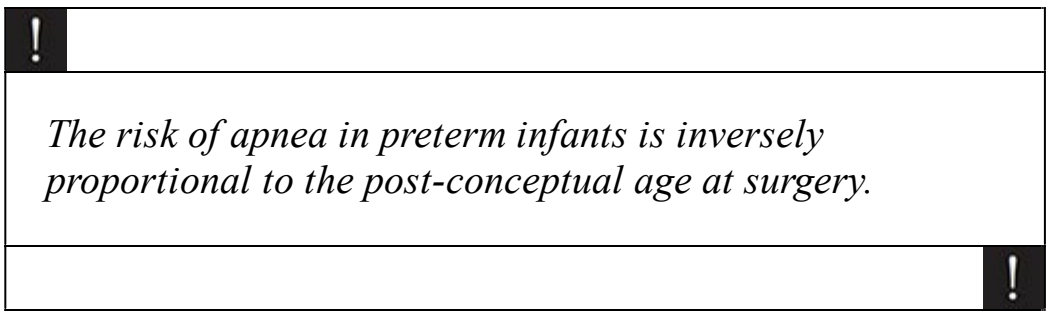
The preoperative history is essential for identifying patients with underlying conditions placing them at potential risk for intraoperative and postoperative complications. This history is an essential part of the handoff communication that should take place between the operating room staff and ICU or ward team assuming care for the patient. It should include prior medications, allergies, chronic medical conditions (eg, asthma, epilepsy), and reactions to any anesthetic agents or procedures. Pertinent information about the intraoperative course should be relayed to the medical team providing postoperative care. This would include: (1) procedure performed; (2) anesthetic agents used; (3) difficulty in airway and ventilatory management; (4) type and dose of opioids administered; (5)

estimated blood loss and transfusions administered; (6) fluids administered; (7) operative findings; (8) any complications; and (9) the presence of tubes, lines, and drains.

III. RESPIRATORY CONSIDERATIONS

A. Postoperative Apnea

Postoperative apnea is defined as the cessation of respirations for ≥15 seconds or the cessation of breathing for <15 seconds when associated with bradycardia or desaturation. Premature infants are at increased risk for apnea (central and obstructive) within the first 48 hours after anesthesia; this usually presents in the first 2 to 12 hours postoperatively and may be due to immaturity of the respiratory center in the brainstem and easy fatigability of the neonatal diaphragm. Apnea is a stress response in neonates and may be associated with inadequate anesthesia and analgesia.



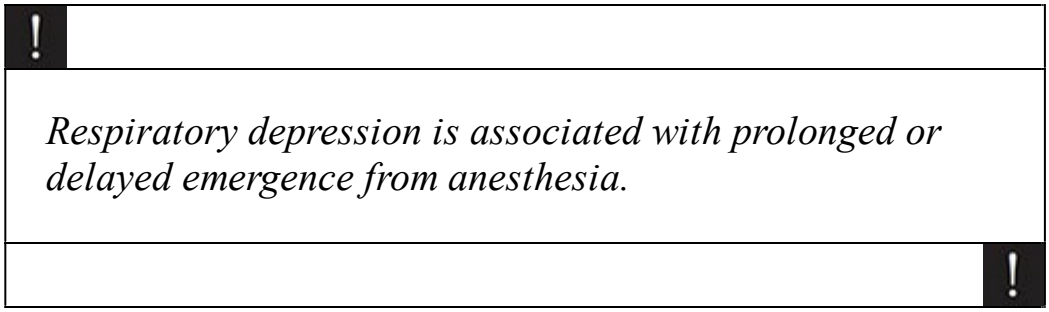
Premature and full-term infants younger than 44 weeks' postconceptual age are at risk for apnea. The age at which this risk disappears continues to be a controversy. Anesthesia should be avoided in the full-term infant younger than 44 weeks' postconceptual age, unless emergent surgery is required. Full-term infants younger than 1 month and premature infants younger than 60 weeks' postconceptual age should be considered at risk for apnea after general anesthesia, and these infants require inpatient monitoring and observation for at least 12 hours after surgery. Use of low-dose aminophylline, nonopioid pain medications, and awake regional anesthesia reduce the risk of postoperative apnea in premature babies.

Anemia is an independent risk factor that increases the chances of postoperative apnea in the neonatal population. It is recommended that infants with a hemoglobin level <10 g/dL should have elective surgery postponed and be given oral iron supplements until the hemoglobin measurement is >10 g/dL. If surgery cannot be delayed, these patients require postoperative inpatient observation and monitoring. Infants with a history or risk of obstructive sleep apnea and those being treated for apnea of prematurity should be observed for 24 hours postoperatively.

B. Respiratory Depression

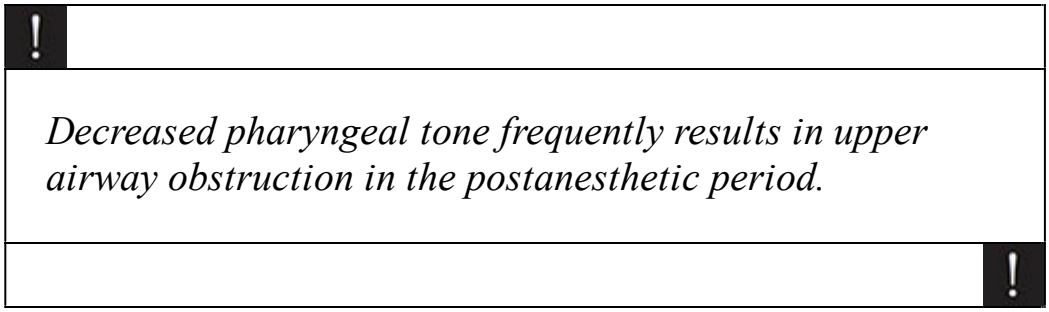
Respiratory depression in the immediate postoperative period is usually caused by opioids, sedatives, residual anesthetic effect, or inadequate neuromuscular blockade reversal. Severe chest or abdominal pain may also cause respiratory splinting with resultant hypoventilation and delay in the

elimination of any inhaled anesthetic agents. If an excessive opioid or benzodiazepine effect is suspected, gradual reversal is warranted with naloxone and flumazenil, respectively. The half-life of these agents is relatively short compared with that of most opiates and benzodiazepines, and the patient should be monitored for recurrence of respiratory depression.

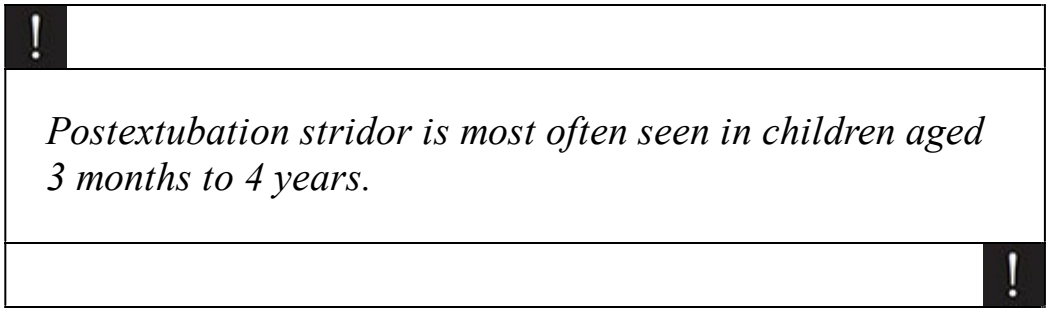


C. Upper Airway Obstruction

Upper airway obstruction from a floppy tongue is frequently encountered in the postoperative period because of a residual anesthetic effect causing pharyngeal hypotonia. Postoperative airway obstruction often manifests as paradoxical respiration or seesaw-type breathing (ie, the chest caves in while the abdomen rises during inspiration, and chest expands with abdomen caving in during exhalation). Prompt recognition and intervention in the form of airway positioning with the jaw-thrust maneuver relieves the obstruction in most cases, although the use of nasal or oral airway adjuncts may be necessary in some. In all situations, the patient must be monitored and the healthcare provider should be prepared to provide a definitive airway and to support oxygenation and ventilation (**Chapter 2**) until the patient is awake or a definitive airway is secured.



Postextubation stridor occurs in about 2% of all postoperative pediatric patients. Etiologies include: subglottic edema arising from the use of oversized endotracheal tubes, traumatic or repeated attempts at intubation, coughing or bucking with the endotracheal tube in place, mechanical ventilation lasting longer than 24 hours, or a change in the position of the endotracheal tube.



The use of appropriately sized endotracheal tubes with a leak <30 cm H_2O decreases the risk of airway trauma. If the patient has evidence of significant stridor and increased work of breathing, nebulized racemic epinephrine/adrenaline (0.25-0.5 mL of 2.25% solution in 2-3 mL of normal saline solution) should be administered. The patient will require observation in a monitored environment for 4 hours because of potential rebound edema. Corticosteroids (dexamethasone 0.5 mg/kg intravenously or intramuscularly, maximum dose 12 mg) should be administered for persistent stridor and signs of upper airway obstruction. Heliox (a mixture of helium and oxygen) may help improve the flow of gases and reduce the work of breathing. Patients who do not respond to these interventions and demonstrate marked increased work of breathing, altered sensorium, desaturations, and ineffective gas exchange should be reintubated. When attempting reintubation in this scenario, use an endotracheal tube that is at least a half size smaller in diameter than the tube used in anesthesia. Patients who are awake and exhibiting stridor may be managed with noninvasive ventilation.

Other possible causes of upper airway obstruction include: laryngomalacia; soft tissue swelling of oropharyngeal structures (eg, tongue); laryngospasm; uncontrollable bleeding from the upper airway; and posterior pharyngeal narrowing after cleft palate repair. Usually, these patients will improve with upright positioning, adequate analgesia, suctioning and clearing of secretions, and control of any active bleeding. When in doubt or if the patient's clinical condition is not improving with these interventions, intubation is indicated.

D. Lower Airway Disease

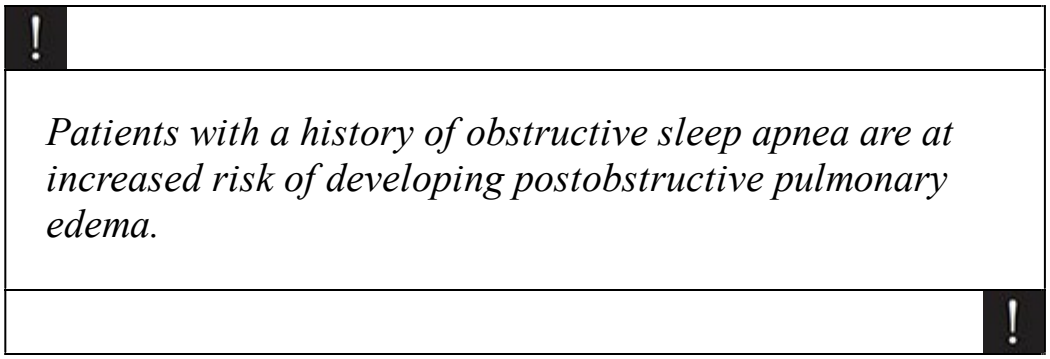
Atelectasis, the most common postoperative respiratory complication, usually occurs in the first 48 hours postoperatively. Alveolar collapse is a result of inhaled anesthetics, high inspired oxygen concentration, and hypoventilation or retained secretions secondary to pain or postoperative narcotics. Postoperative atelectasis can be prevented by continued airway clearance techniques and age-appropriate breathing exercises, such as incentive spirometry or a positive expiratory pressure device for older children, and by encouraging younger children to blow bubbles or a pinwheel. Early ambulation also promotes deep breathing and reduces the incidence of atelectasis.

Hospital-acquired pneumonia is the second most common nosocomial infection and is predominantly seen in ventilated, postoperative, and critically ill patients (**Chapter 7**). The etiology of hospital-acquired pneumonia is microaspiration of bacteria colonizing the oropharynx or gastrointestinal tract. Pneumonia occurs after 48 hours in the postoperative patient who does not have an antecedent infection. The main preventive interventions are elevating the head of the bed 30° to 45° (if no contraindications), early ambulation, and encouraging pulmonary toilet.

E. Pulmonary Edema

Pulmonary edema is usually rare, unless the patient is known to have congestive heart failure or received an excessive amount of IV fluids intraoperatively. Postobstructive pulmonary edema (POPE), previously known as *negative pressure pulmonary edema*, occurs after the acute relief of chronic severe upper airway obstruction. This may be seen in children following an adenotonsillectomy for obstructive sleep apnea. It also can occur after acute postextubation

obstruction, such as laryngospasm, in which the patient makes forceful inspiratory efforts against a closed glottis.



Patients with a history of obstructive sleep apnea are at increased risk of developing postobstructive pulmonary edema.

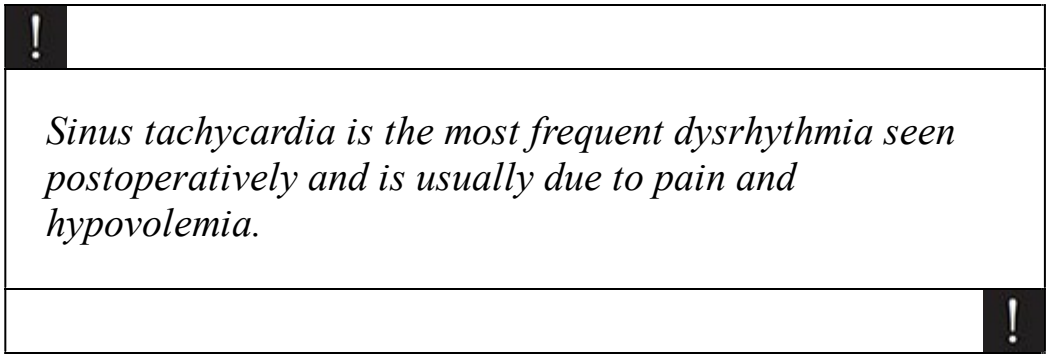
Management of pulmonary edema is supportive, with mild cases requiring oxygen and possibly a dose of IV diuretic (furosemide, 1 mg/kg). Patients in severe respiratory distress may be managed with noninvasive mechanical ventilation or intubation and adequate positive end-expiratory pressure. Usually, pulmonary edema in these cases rapidly resolves with appropriate management.

IV. HEMODYNAMIC CONSIDERATIONS

A. Dysrhythmias

Children tend to be healthy, and postoperative abnormal electrocardiogram and arrhythmias are uncommon in the absence of an underlying cardiac pathology or electrolyte disturbance. An abnormal heart rate is the most frequent dysrhythmia, and sinus tachycardia is the most common.

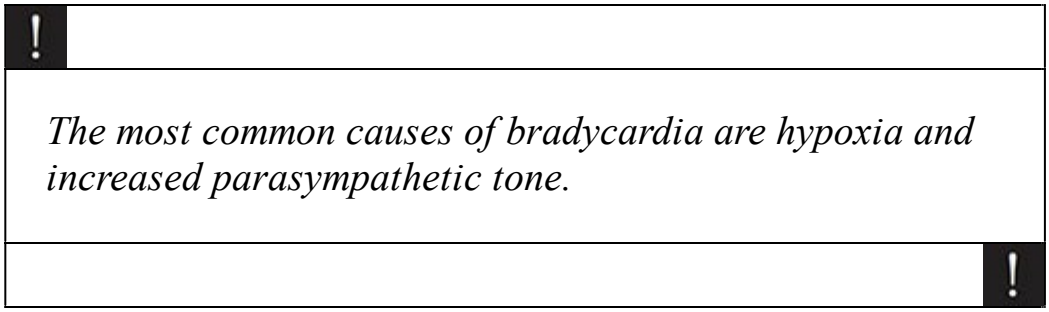
Sinus tachycardia rarely needs specific therapy, but its underlying etiology must be determined. Tachycardia may be an early indicator of compensated shock, with under-resuscitated hypovolemia as an underlying cause. Patients may exhibit cool, clammy extremities, thready pulses, delayed capillary refill, and decreased urine output. Hypotension is a late sign indicating decompensation of the physiologic state. Prompt fluid resuscitation is of paramount importance to prevent further deterioration of shock. Other factors that may result in sinus tachycardia include emergence agitation, pain, anemia, anxiety, hyperthermia, hypercarbia, and the presence of parasympathomimetic agents (such as atropine and glycopyrrolate) and incomplete neuromuscular blockade reversal.



Sinus tachycardia is the most frequent dysrhythmia seen postoperatively and is usually due to pain and hypovolemia.

Bradycardia requires immediate attention in infants, because these patients depend on the heart rate

for cardiac output. The most life-threatening cause of bradycardia is hypoxia; thus, administering oxygen and providing assisted ventilation are paramount in the initial management. Tracheal suctioning, nasogastric tube insertion, or the application of ophthalmic pressure can result in increased parasympathetic tone, a common cause of bradycardia in infants.



Myocardial ischemia, electrolyte imbalances, and vagal nerve stimulation are causes to be ruled out with arrhythmias following cardiac surgery. Patients who have undergone cardiac surgeries and who are at risk of dysrhythmia should receive continuous electrocardiogram monitoring in the immediate postoperative period.

B. Abnormal Blood Pressure

Hypertension in children rarely has a pathologic etiology and, like tachycardia, usually does not require specific therapy, but the underlying cause must be determined. Hypertension with associated tachycardia is usually due to increased agitation, anxiety, pain, or parasympathetic effect. When accompanied by a normal or decreased heart rate, this may be a sign of hypothermia, increased intracranial pressure, or the effect of α-adrenergic therapy, situations in which antihypertensives are not indicated. Antihypertensive therapy is appropriate in patients who are known to have chronic renal disease with preexisting hypertension.

Hypotension in the postoperative patient should be considered a medical emergency and is almost always secondary to hypovolemia. This also may be seen during rapid rewarming of patients in the postoperative period. However, the most common cause of hypotension is volume depletion. If the patient does not respond favorably to volume expansion, sepsis, cardiac dysfunction, or anaphylaxis is highly likely. In these situations, vasopressor and/or inotropic support may be necessary with subsequent monitoring in ICU setting.

V. NEUROLOGIC CONSIDERATIONS

Case Study

A 10-year-old boy with a history of postmeningitic hydrocephalus and placement of an indwelling ventriculoperitoneal shunt presents with recurrent vomiting, headache, fever, and lethargy. A

computed tomography scan of the head shows an increase in the ventricular size. The patient underwent externalization of the shunt because of a concern of infection. You are called to the bedside 3 hours after the surgery because the patient exhibits lethargy and is nonresponsive. On physical examination, he is obtunded with a Glasgow Coma Scale (GCS) score of 10. Vital signs are as follows: temperature, 38°C (100.4°F); respiratory rate, 23 breaths/min; heart rate, 110 beats/min; and blood pressure, 110/72 mm Hg.

Detection

– What would you want to know about his preoperative and intraoperative course?

– What are the possible causes for altered mental status in the postoperative period?

Intervention

– What laboratory tests or other studies should be ordered?

– What would be the immediate interventions?

– When would any imaging studies be considered?

Reassessment

– How would you reassess the patient?

– What are the potential complications after neurological surgery?

Effective Communication

– What should be communicated in the handoff between the operating room team and the ward team assuming care for the patient?

Teamwork

– Who else might need to be involved in the care of this patient?

– Where is the best place to monitor or manage the patient?

A. Altered Consciousness

Altered consciousness poses a diagnostic dilemma for the anesthesiologist and the medical team managing the patient postoperatively. This is a common reason for unanticipated admission to the pediatric ICU from the recovery room. The causes for this can usually be traced to prolonged anesthetic effect along with other less common etiologies (**Table 19-1**).

Table 19-1	CAUSES OF ALTERED CONSCIOUSNESS IN THE POSTOPERATIVE PERIOD

- Prolonged anesthesia or delayed emergence due to residual anesthetic effect

- Prolonged neuromuscular blockade
- Hypothermia
- Metabolic and electrolyte imbalances
  –Hypoglycemia
  –Diabetic ketoacidosis
  – Hypokalemia → severe muscular weakness
  –Hypernatremia and hyponatremia
  – Hypothyroidism
- Hypercarbia secondary to hypoventilation
  – Narcosis with markedly elevated CO_2 levels
- Neurologic events
  –Postictal state
  –Embolic event
  –Hypoxic-ischemic injury
  – Increased intracranial pressure

1. Residual Anesthetic Effect

The most common anesthetic agents used in surgery are inhalational anesthetic agents, the elimination of which is dependent on minute ventilation, cardiac output, drug solubility, and the depth and duration of anesthesia. The residual effect of anesthetic agents could prolong the patient's stay in recovery room or necessitate an admission to pediatric ICU, especially if the patient continues to require mechanical ventilation. Excessive opioid administration is a cause of respiratory depression, and this must be evaluated in the patient who fails to awaken. A judicious trial of naloxone could reverse the excessive opioid effect but also the analgesic effect, precipitating pain, agitation, hypertension, and uncontrollable behavior. If there is no improvement in the patient's sensorium after naloxone administration, then other etiologies must be sought.

!

Management of the airway and ventilation until the residual anesthetic effect abates is preferable to the acute reversal of analgesia.

!

2. Prolonged Neuromuscular Blockade

Neuromuscular blocking agents can be divided into two types: depolarizing and nondepolarizing. Succinylcholine is a depolarizing agent that noncompetitively binds to the acetylcholine receptors of the motor endplate. It then diffuses from the receptor and is metabolized by hepatic and plasma

pseudocholinesterase. Prolonged blockade after administration of succinylcholine is most commonly caused by an abnormal genetic variant of pseudocholinesterase. Hepatic dysfunction and hypermagnesemia could also prolong the paralytic effect of succinylcholine. The patient is managed with supportive ventilation until the effects of neuromuscular blockade (NMB) have dissipated.

Nondepolarizing neuromuscular blockers, such as pancuronium, rocuronium, vecuronium, and cisatracurium, competitively inhibit acetylcholine receptors, causing muscular paralysis. This type of agent can be reversed or antagonized by administering an inhibitor of acetylcholinesterase (neostigmine, edrophonium, and pyridostigmine); this results in elevated synaptic concentrations of acetylcholine, which then competitively displace the neuromuscular blocking agent from the receptor. Due to the muscarinic effects of these reversal agents, atropine (0.02 mg/kg IV) or glycopyrrolate (0.01 mg/kg IV) should be given concomitantly to prevent bradycardia. Sugammadex is capable of reversing any depth of neuromuscular blockade induced by rocuronium or vecuronium. It forms a complex with NMB agents in the plasma, reducing the amount available to act on the nicotinic receptors. It acts within 3 minutes and has fewer side effects than neostigmine. Pediatric use of this drug is unavailable in the United States.

Common causes of failure in reversal of nondepolarizing agents are given in **Table 19-2**.

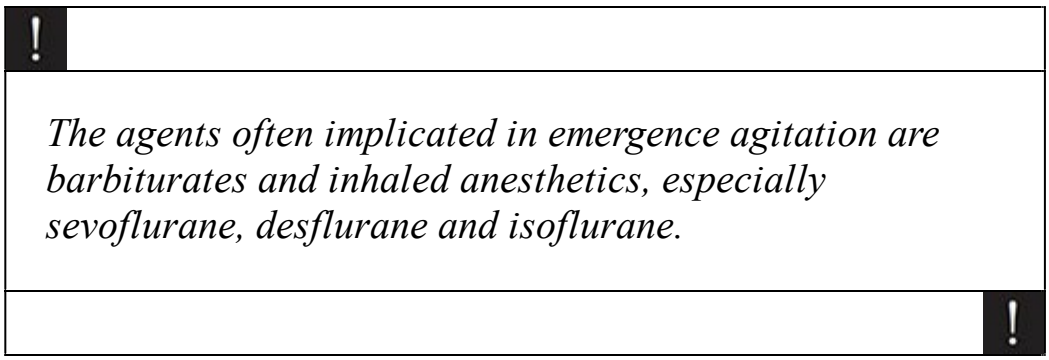
Table 19-2	FAILURE OF REVERSAL OR RECURRING BLOCKADE BY NONDEPOLARIZING AGENTS

- Respiratory acidosis
- Hypokalemia
- Hypermagnesemia
- Hypothermia
- Drug interactions
  - –Antibiotics: aminoglycosides, tetracyclines, polymyxins, lincomycins
  - –High-dose corticosteroids
  - –Calcium channel blockers
  - –Antiarrhythmics (quinidine, procainamide)
  - – Alkylating cytotoxic agents
- Myasthenia gravis
- Muscular dystrophies and myotonia

Patients with prolonged NMB will not be able to breathe or move but will retain autonomic and ocular reactivity. They can be evaluated using a peripheral nerve stimulator with the administration of a train-of-four electrical impulses, while observing for corresponding twitches. Usually, the fourth twitch is abolished when >75% of the acetylcholine receptors on the motor endplate are blocked. Adults can still breathe effectively when <75% of the receptors are blocked, but children have decreased functional residual capacity and a higher incidence of postextubation airway obstruction. Other indicators of adequate reversal of NMB include the patient's ability to lift the head for longer than 5 seconds, to exhibit a purposeful handgrip, and to achieve a sustained leg raise.

B. Emergence Agitation

Emergence agitation (EA) is a well-described phenomenon typically occurring within 30 minutes of postoperative recovery. Patients with EA exhibit a dissociated state of consciousness in which they are inconsolable, irritable, and uncooperative; they typically are thrashing, crying, moaning, or incoherent within the first few postoperative hours. Often these children do not recognize or identify familiar objects or people. The incidence of EA in children is greater than in adults (12% vs. 5.3%), has no gender predilection, and is most commonly seen in those between ages 2 and 5 years. These patients pose a risk of harming themselves and often will require restraints, comforting by caregivers, and reduced environmental stimuli (eg, dark quiet room). EA most often resolves spontaneously, but sometimes "rescue" medication (analgesics, benzodiazepines, or hypnotics) is required. The agents that have been used successfully are propofol, dexmedetomidine/clonidine, and fentanyl; however, EA may recur after the effects of these agents have worn off. Other causes for agitation should be ruled out, such as pain, hypoxia, hypercapnia, hypoglycemia, and anxiety.


!
The agents often implicated in emergence agitation are barbiturates and inhaled anesthetics, especially sevoflurane, desflurane and isoflurane.
!

VI. PAIN MANAGEMENT

Case Study

An obese 4-year-old girl underwent tonsillectomy and adenoidectomy for recurrent tonsillitis and obstructive sleep apnea. She was admitted for overnight observation. You were notified that she is constantly crying and is agitated. Examination reveals the following: heart rate, 130 beats/min; respiratory rate, 40 breaths/min; temperature, 38.6°C (101.4°F); and blood pressure, 100/70 mm Hg. Her body mass index is 32.

Detection

– What would you want to know about her preoperative and intraoperative course?

– What are the possible reasons for the patient's agitation?

Intervention

– What would be the best approach to her pain management?

– How would you monitor the patient postoperatively?

– What would be your immediate intervention?

Reassessment

– How would you assess the effectiveness of the patient's pain medication?

– What are the potential complications for each medication?

– What are the potential causes of continued postoperative fever?

Effective Communication

– What should be communicated in the handoff between the operating room team and the ward team assuming care for the patient?

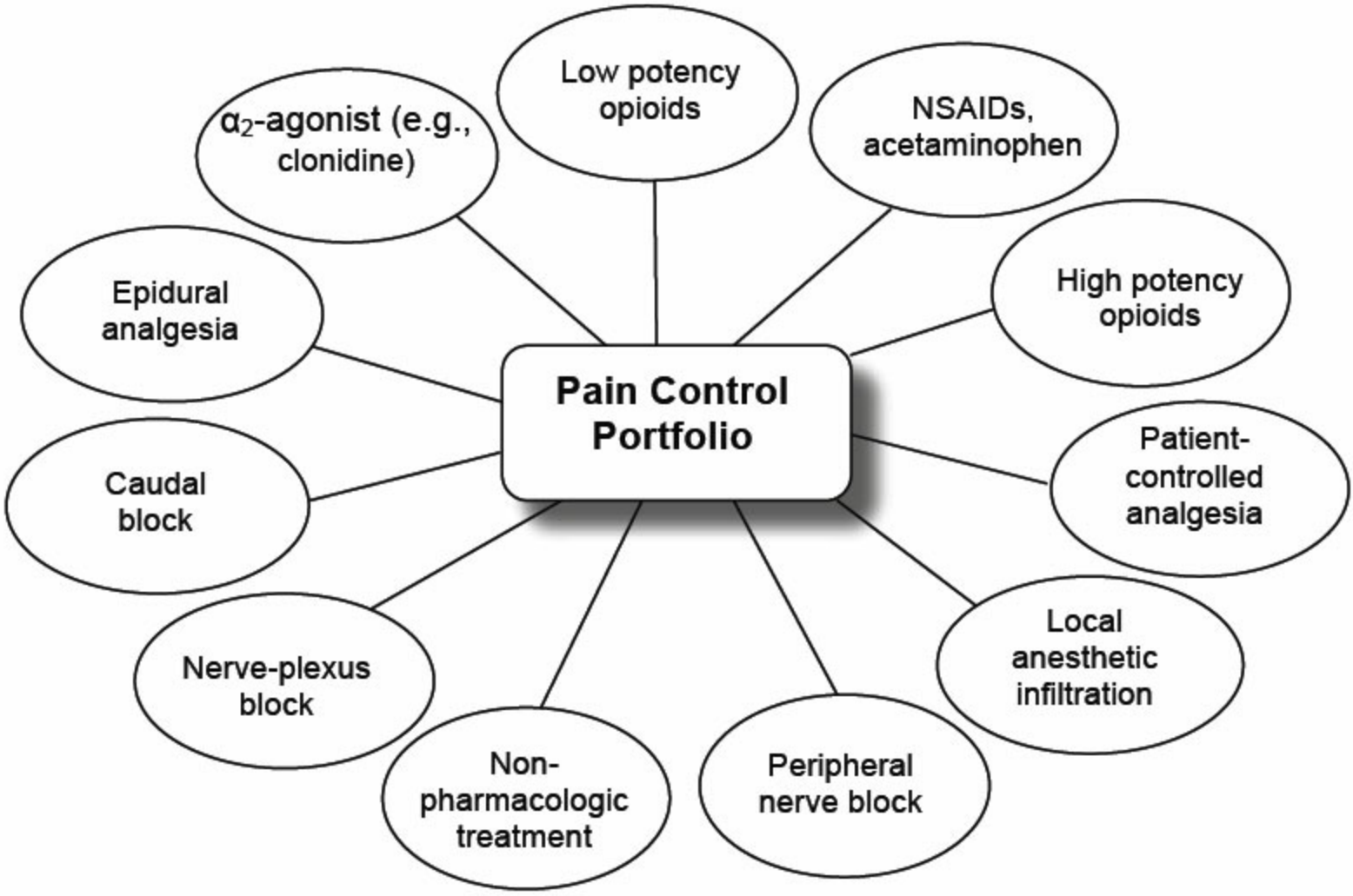
Teamwork

– What are the roles of other healthcare providers in the postoperative management of this patient?

Children of all ages, including premature neonates, experience pain. Approximately 40% to 75% of children will experience moderate to severe pain in the first 24 hours after surgery. Pain management requires a multimodal approach to optimize the analgesic effect and minimize any side effects. Strategies are determined by the type of surgery, anesthetic technique used, expected duration of the recovery period, and anticipated severity of postoperative pain.

Nociception is the complex process whereby noxious stimuli are sensed, transformed, transmitted, modified, and eventually perceived as pain. The multimodal approach to pain management utilizes pharmacologic and nonpharmacologic therapies to target multiple steps in the process. Use of a combination of analgesics and adjuncts is more effective in pain control than an analgesic that targets a single step in the nociceptive pathway (**Figure 19-1**). This strategy also provides the benefit of reducing opiate use and side effects.

Figure 19-1. Multimodal Therapy for Postoperative Pain Management



NSAIDs, nonsteroidal anti-inflammatory drugs

A. Pain Assessment

Pain assessment in children can be difficult; their responses to pain are age-specific and often difficult to distinguish from agitation and anxiety. Many children respond to pain by withdrawing from their surroundings and withholding information out of fear of further painful interventions. This can be misinterpreted by the medical staff as a lack of pain. Validated, developmentally appropriate tools have been developed to objectively assess pain in neonates, infants, cognitively impaired children and ventilated patients (**Table 19-3**). The child between 3 and 7 years of age can report pain using the FACES Pain Rating Scale. Older children can use verbal or adult visual analog scales. Consistent application of a pain assessment tool that is specific for the patient population and situation is extremely important.

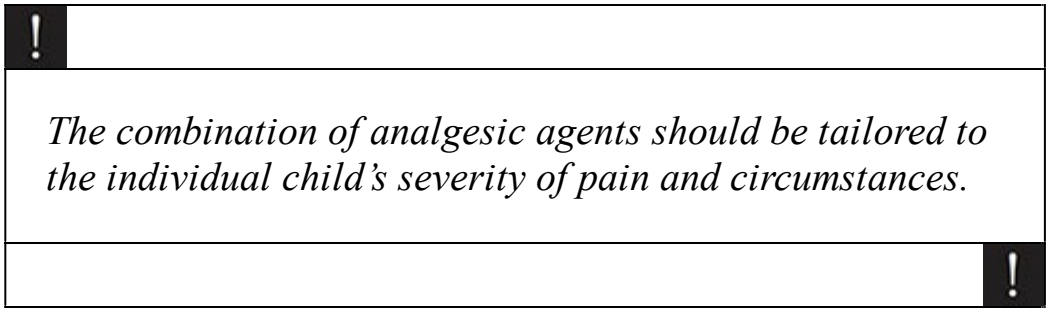
Table 19-3 PEDIATRIC PAIN ASSESSMENT TOOLS

Pain Scale	Age	Type	Scale Parameters	Uses
CRIES	32-60 weeks	Behavioral and physiologic parameters	Crying, requires increased O_2, increased vital signs, expression, sleepiness	Acute pain, procedural and surgical pain
PIPP (premature infant pain profile)	Preterm and term infants	Behavioral and physiologic	Gestational age, behavioral state, heart rate, O_2 saturation, facial expression	Procedural pain

		parameters		
FLACC	<3 years	Behavioral parameters	Face, legs, activity, cry, consolability	Acute pain, surgical pain
r-FLACC	Nonverbal	Behavioral parameters	Face, legs, activity, cry, consolability	Acute pain, surgical pain
Faces scales (eg, Wong-Baker FACES)	3-12 years	Subjects rate pain	Happy to saddest face with 0-10 score	Acute pain, surgical pain
Visual analog scale (VAS)	>7 years	Subjects rate pain	Horizontal 10-cm line; 0 = no pain, 10 = most pain	Acute pain, surgical pain, chronic pain
Numerical scale	>7 years	Subjects rate pain	Integers from 0-10; no pain to most pain	Acute pain, surgical pain, chronic pain

B. Multimodal Strategy of Pain Management

A variety of analgesic drugs can be used in the postoperative pain management of children, including nonopioids, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, and regional analgesia.



The combination of analgesic agents should be tailored to the individual child's severity of pain and circumstances.

Single-drug therapy with opioids has been the traditional method by which pediatric postoperative pain was managed. This often resulted in inadequate pain control and the common occurrence of adverse drug effects such as nausea, vomiting, pruritus, constipation, urinary retention, and respiratory and central nervous system depression. The dependence on a single analgesic often results in dosing delays, inadequate dosing, and failure to recognize an individual's pain.

A multimodal analgesia plan (**Figure 19-1**) provides a balanced approach that targets several of the pathways of nociception and encompasses combinations of the following therapies: (1) acetaminophen/paracetamol and NSAIDs acting on the periphery; (2) regional anesthetic blockade of peripheral nerves, nerve roots, or the spinal cord; (3) opioids acting centrally; and (4) nonpharmacological treatments incorporating distraction, and cognitive-behavioral and psychological interventions. This multimodal strategy can minimize the occurrence of adverse drug events by using smaller dosages of each analgesic while still achieving equivalent or superior pain control compared to opioid-only therapy. The weaknesses of opioid-only therapy can be often be resolved by scheduled dosing of acetaminophen or NSAIDs in the first 24 to 72 hours postoperatively. Other choices include patient-controlled analgesia (PCA) and long-acting regional analgesic techniques. Choices should be tailored to the individual patient's circumstances and pain level.

Use of nonopioid analgesics is becoming more popular in the pediatric postoperative setting, avoiding or reducing the side effects of opioids. Scheduled acetaminophen and NSAIDs dosing has

been proved to be as effective as opioids and sometimes provides improved pain control after surgeries like tonsillectomy and adenoidectomy, spinal fusion, inguinal herniorrhaphy, and pediatric palatoplasty. Scheduling the nonopioid medication in the first 24 to 72 hours postoperatively results in reduced opioid administration for breakthrough pain. Analgesic and antipyretic effects of IV acetaminophen are comparable to those achieved via the oral or rectal route, but the onset of action is faster (onset in 15 minutes with peak effect within 1 hour). IV acetaminophen is approved by the US Food and Drug Administration for children older than 2 years and may be administered at the start of an operative procedure to reduce intraoperative opioid use. Postoperatively, IV acetaminophen use should be reserved for when the oral and rectal routes are either not available or contraindicated. Scheduled ibuprofen after tonsillectomy should be used cautiously due to the risk for increased postoperative bleeding.

A stratified method for the use of analgesics in pain management is based on the severity of pain experienced and closely follows the Analgesic Ladder model of cancer pain management developed by the World Health Organization. In this approach, nonopioids, including acetaminophen and NSAIDs (eg, ibuprofen) can be used for mild pain. Lower potency opioids are often used in combination with nonopioids for moderate pain; for severe pain, IV opioids (morphine, fentanyl, hydromorphone) are utilized in combination with potent IV NSAIDs (ketorolac, 0.5 mg/kg IV every 6 h) or scheduled acetaminophen (15 mg/kg oral/rectal/IV every 6 h or 12.5 mg/kg oral/rectal/IV every 4 h).

Clonidine is an α_2-adrenergic agonist and a useful adjunct in the postoperative pain management plan. When given preoperatively, it can reduce the postoperative opioid requirement. It also is a local anesthetic adjunct and can prolong and improve analgesia in peripheral nerve blocks and caudal, epidural, and spinal anesthesia, thereby reducing the need for opiates. Dexmedetomidine, another α_2-adrenergic receptor agonist, acts centrally and has 8 times the affinity for the α_2-receptor as clonidine. It provides good sedation and analgesic benefits and can potentially reduce the need for opioids. The other advantage to dexmedetomidine is that it is associated with minimal respiratory depression.

An anesthesia or pain service consultation is highly recommended if a patient is expected to have severe, difficult-to-control, or refractory pain postoperatively.

C. Patient-Controlled Analgesia

Typically, opioids are administered after patients complain or are noted to be in pain. This leads to a vicious cycle of pain, followed by delayed administration of an analgesic, with the resultant risk of persistent pain or increased sedation. It places the patient at risk for the side effects of opioids with inadequate pain relief. This cycle can be broken by the use of PCA, an excellent delivery system for the control of moderate to severe pain when used with adequate monitoring. PCA can be used in patients as young as 5 years (with proper education) and applied routinely in children 7 years or older. Any child who can play video games or operate a smartphone may use a PCA device. The goal is twofold: (1) maintain therapeutic opioid levels to insure adequate analgesia; and (2) utilize a system that is rapidly responsive to the patient's analgesic needs. PCA has high rates of patient, parent, and staff satisfaction. The PCA regimen usually employs an opioid that can be dosed on demand and/or continuously (basal infusion) [**Table 19-4**].

Any child who can play video games can operate the PCA device.

Table 19-4	PATIENT-CONTROLLED ANALGESIA DOSING REGIMEN			
Opioid Drug	**Demand Dose (µg/kg)**	**Lock-out Interval (min)**	**Basal Infusion (µg/kg/h)**	**1-Hour Limit (µg/kg)**
Morphine	20 (adult dose: 1000 µg)	8-10	0-20	100 (adult dose: 2000 µg/h)
Fentanyl	0.5 (adult dose: 50-100 µg)	6-8	0-0.5	2.5 (adult dose: 50-100 µg/h)
Hydromorphone	4 (adult dose: 200 µg)	8-10	0-4	20 (adult dose: 200 µg/h)

Data taken from Landsman IS, Vustar MV, Hays SR. Pediatric anesthesia. In: Grosfeld JL, O'Neill JA, Coran AG, Fonkalsrud EW, eds. *Pediatric Surgery*. 6th ed. Philadelphia, PA: Mosby Elsevier; 2006:221-256.

Continuous basal infusions should only be administered under close cardiorespiratory monitoring; they should be avoided in the opiate-naive patient except in special circumstances, such as posterior spinal fusion. Basal infusions may facilitate a normal sleeping pattern in the postoperative adult patient, who is awoken by pain and then self-administers a dose of opioid. However, no evidence supports this in children, and the risk of adverse respiratory events is increased with continuous opioid infusions. Because of this risk, the routine use of basal infusions is not recommended for nonintubated patients.

Demand dosing is often used as this ensures a margin of safety, while providing adequate analgesia and minimizing opioid side effects. The minimum time interval between demand doses of narcotics is called the *lockout time*, and no drug is delivered during that time no matter how often the button is pressed. This is intended to prevent narcotic accumulation. A total maximum dose (1- to 4-hour limits) is programmed into the pump to prevent overdosing. To optimize patient safety and prevent adverse events, oxygen saturation, respiratory rate, heart rate, blood pressure, end-tidal CO_2, pain, and level of consciousness must be monitored. Rescue orders should be part of the PCA treatment regimen and naloxone (1-2 µg/kg every 5 minutes to a total of 10 µg/kg) is prescribed to reverse the untoward adverse opioid effect of respiratory depression. Severe breakthrough pain is treated with additional IV opioids (same narcotic medication used in the pump) and administered every 3 hours as needed. Pruritus induced by opioids can be treated with nalbuphine (50 µg/kg IV every 4 hours) and in the event of severe pruritus, a naloxone infusion (0.25 µg/kg/h) can be used. PCA can be utilized in conjunction with acetaminophen/paracetamol or NSAIDs to reduce the amount of opioid consumption and side effects. Some practitioners avoid short-acting oral opioids in patients already using PCA, but occasionally substitute a slow release oral drug for a basal infusion.

PCA by proxy allows the parent or nurse to press the PCA button to deliver narcotics to children younger than 6 years or to those with developmental delay. However, different parameters must be set on the PCA pump to prevent accidental overdose by caregivers, and the patient's environment should be monitored. PCA by proxy has the same incidence of adverse events as PCA by patient, but the need for rescue interventions is greater.

D. Epidural Analgesia

Continuous epidural analgesia (CEA) is an invaluable method of treating severe postoperative pain in neonates, infants, and children. CEA decreases the stress response and duration of hospital stay, and may improve outcomes in particular pediatric groups. The epidural catheter is inserted via the caudal approach in infants or inserted into the low thoracic or lumbar interspace of L5 and S1. The epidural catheter is threaded to a thoracic or high lumbar level, with the catheter tip placed adjacent to the center of the dermatomes to be blocked. This ensures adequate pain control and a continuous infusion of local anesthetic with the added benefit that an opioid can be delivered to provide continuous pain relief. The combined effect of local anesthetics and opioids is synergistic, so lower doses of each agent can provide adequate analgesia.

A common problem is inadequate pain control due to inaccurate catheter placement, catheter problems (kinking, leaking, dislodgement, breakage), or an insufficient infusion rate or analgesic dose. The possible complications of CEA include: (1) local anesthetic toxicity; (2) motor block as a direct effect of local anesthetics or a late sign of compartment syndrome or epidural hematoma; (3) epidural abscess; (4) adjuvant opioid side effects; (5) inadvertent intrathecal administration; and (6) failure to achieve complete block. Reducing the concentration or infusion rate of the local anesthetic or using medication to counteract the opioid-induced side effects can usually manage these complications. The patient is routinely monitored for motor block, dermatome block, and fever, along with changes in hemodynamic and respiratory parameters. Vigilance is needed in monitoring for respiratory depression caused by the dermatome block being too high or, more frequently, because IV or intramuscular opioids are used to supplement the epidural opioid.

Patient-controlled epidural analgesia combines the benefits of CEA and PCA and allows the timely response to pain exacerbation.

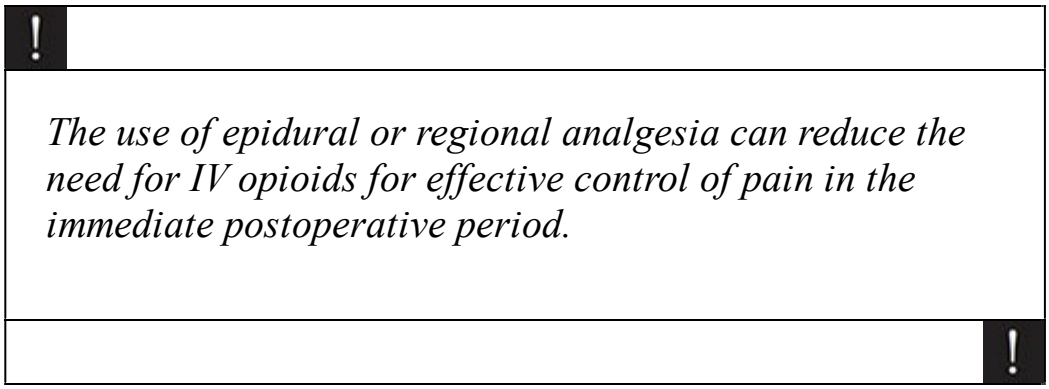
E. Regional Anesthesia

Regional anesthesia techniques are becoming popular in pediatric postoperative pain management. With the aid of ultrasound and nerve stimulators, regional anesthesia has improved the success of nerve blocks. These techniques use local anesthetics to block the afferent pathways between nociceptors and the central nervous system and include local infiltration, peripheral nerve blocks, nerve-plexus blocks, and epidural or intrathecal nerve blocks. The anesthetic agent is administered as a single injection or by continuous infusion. Often long-acting local anesthetics are used. Examples of nerve blocks include penile, femoral, sciatic, axillary, ankle, intercostal, and caudal blocks.

Regional anesthesia can be advantageous in patients who exhibit increased sensitivity to the side effects of opioids, which often occur in neonates and young children. It can also lower the risk of

adverse effects frequently encountered with parenterally administered opioids, such as nausea, vomiting, sedation, and respiratory depression. The use of long-acting local anesthetics can reduce the need for high-potency opioids and provide hours of pain relief in the immediate postoperative period when the severity of pain is usually at its greatest.

The most common regional anesthesia used in children is the caudal block. In this technique, the epidural space is accessed via the sacral hiatus, and a single injection of 1 mL/kg of local anesthetic is administered. This block can be performed with the aid of ultrasound guidance, if available. Its success rate is high and complication rate low. It usually provides analgesia below the umbilicus and, with the use of only a local anesthetic, the patient is pain-free for several hours. In general, children do not exhibit hemodynamic instability or urinary retention. The addition of opioids will prolong analgesia but increase the risk of side effects. This technique usually works well when severe pain is anticipated only for the first 24 hours postoperatively.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The use of epidural or regional analgesia can reduce the need for IV opioids for effective control of pain in the immediate postoperative period.
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VII. FLUID AND ELECTROLYTE CONSIDERATIONS

A 10-year-old girl was admitted with fever, abdominal pain, nausea, and tachycardia. She underwent emergent laparoscopic appendectomy and pelvic abscess drainage for perforated acute appendicitis. She was moved to the postoperative floor with a nasogastric tube, 2 peritoneal drains, and a urinary catheter in place. She appears pale. Her vital signs include: heart rate, 140 beats/min; respiratory rate, 40 breaths/min; and blood pressure, 100/70 mm Hg. She exhibits shallow rapid breathing. The abdomen is tense with voluntary guarding. The nasogastric tube has produced 100 mL of bilious drainage; the peritoneal drain has produced 300 mL of serosanguinous drainage.

Detection

– What is the most likely etiology of the tachycardia?

– What information you would like from the preoperative and intraoperative periods?

Intervention

– How will the fluid hydration be managed?

– What type of fluid would you give this patient?

– What would be the best approach to her pain management?

Reassessment

– How would you assess the effectiveness of the patient's fluid resuscitation?

– What parameters will be used to monitor the patient?

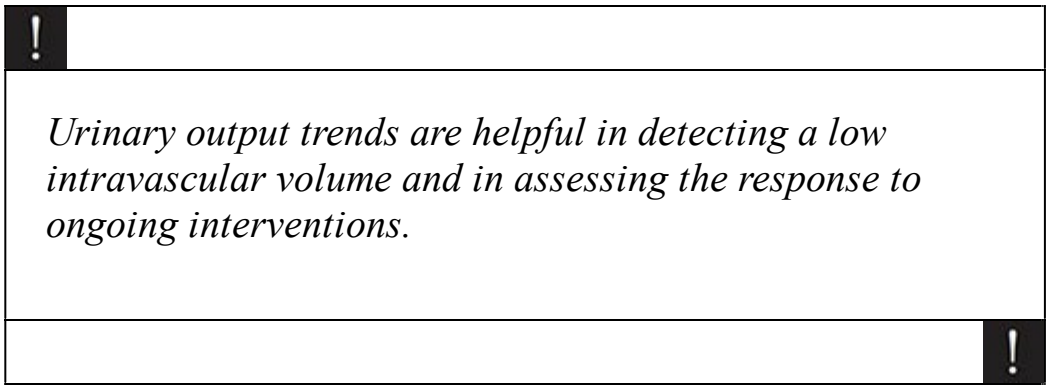
Effective Communication

– What should be communicated in the handoff between the operating room team and the ward team assuming care for the patient?

Teamwork

– Who else might need to be involved in the care of the patient?

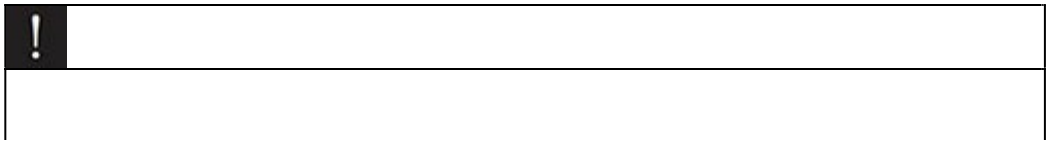
Fluid and electrolyte deficits are some of the most commonly encountered problems in the care of the pediatric surgical patient. These deficits are usually caused by ongoing loss of gastrointestinal fluids (vomiting, nasogastric tubes, and surgical drains), cerebrospinal fluid loss through external ventricular drains, and sequestration of fluids (peritonitis, ileus, burns). Small infants can have extraordinary insensible fluid losses, especially if they are placed under radiant warmers or receiving phototherapy. In these conditions, fluid requirements are increased by 10% to 20%. Pulmonary water loss is negligible when patients are mechanically ventilated due to humidity in the circuits.



Urinary output trends are helpful in detecting a low intravascular volume and in assessing the response to ongoing interventions.

The clinical findings in children will depend on the severity of vascular volume depletion and may manifest as irritability that progresses to apathy and lethargy, increased heart rate, mottled and cold extremities, and decreased urinary output. Hypotension is a late finding in shock. Laboratory evaluation will usually show increased levels of blood urea nitrogen, with or without elevation in creatinine, and hemoconcentration with an inappropriately elevated hematocrit level. In severe volume depletion with significant compromise in oxygen delivery, a metabolic acidosis and increased blood lactate level can be seen.

Children anticipated to have large perioperative fluid shifts or losses and who require large-volume fluid resuscitation should be monitored invasively with continuous measurement of central venous pressure and arterial pressure. An indwelling urinary catheter should be utilized.



Postoperative fluid correction = acute correction of shock + remaining fluid deficit + maintenance fluid + ongoing losses.

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After the acute correction of shock, fluid management should provide for the correction of the remaining deficit, maintenance fluid, and replacement of ongoing losses. In the critically ill postoperative patient, it is safe to assume that any ongoing losses are more likely to be isotonic than hypotonic in composition. It is also safe to assume that the stressed postoperative patient will experience hemodynamic and nonhemodynamic stimuli for the release of antidiuretic hormone. In this setting, isotonic fluids in the early postoperative period decrease the risk of hyponatremia and potential neurologic complications. The patient should be assessed frequently to check hydration and circulation, to determine serum electrolytes (every 6 to 8 hours), and to adjust electrolyte delivery in the fluids accordingly.

The neurosurgical or head-injured patient who develops the syndrome of inappropriate secretion of antidiuretic hormone poses a special circumstance. Such patients are usually euvolemic or have a mild excess of total body water and varying degrees of hyponatremia. They require the provision of maintenance sodium and fluid restriction to 50% of maintenance; the goal is to increase serum sodium at a rate of less than 0.5 mEq/L/h or 12 mEq/L/d (**Chapter 8**).

VIII. POSTOPERATIVE NAUSEA AND VOMITING

Nausea and vomiting – distressing aspects of the postoperative period for patients, parents, and healthcare professionals – are responsible for the adverse effects of increased rates of hospital readmission, delay of hospital discharge, postoperative bleeding, aspiration, wound dehiscence, dehydration, and delay of enteral nutrition. Postoperative nausea and vomiting (PONV) are caused by numerous factors, including the emetogenic effects of inhaled anesthetics, pain, and opioids. The incidence of postoperative vomiting (POV) in children is estimated to be twice that seen with PONV in adults. Children younger than 3 years have an incidence of POV of 22% to 40%, whereas older children have an incidence of 42% to 51%. The risk factors for pediatric POV are: (1) anesthesia lasting longer than 30 minutes; (2) age ≥3 years; (3) strabismus surgery; and (4) history of POV, PONV, or motion sickness in the patient, parents, and siblings. The incidence of POV was 9%, 10%, 30%, 55%, and 70% for these risk factors, respectively. Therefore, patients with a higher risk of POV can be identified and treated with a tailored multistep prophylactic and therapeutic antiemetic plan.

PONV is managed using multimodal therapy because of the limited effects of using single-antiemetic drug therapy. An algorithm for the management of POV/PONV (**Table 19-5**) starts with identifying patients with moderate to severe risk, then preemptively employing 2 or more different classes of antiemetics for prophylaxis. With the addition of each antiemetic drug, the risk of PONV decreases by 30%. This is augmented by avoiding or minimizing the use of inhaled anesthetics and postoperative opioids with the use of total IV anesthesia (eg, propofol), regional anesthesia, and NSAIDs.

Table 19-5	MULTIMODAL APPROACH TO POSTOPERATIVE NAUSEA AND VOMITING

- Evaluate risk
- If moderate or severe risk:
  - Reduce baseline risks
    - Avoidance/minimization of inhaled anesthetics, neostigmine, nitrous oxide, and postoperative opioids
    - Total IV anesthesia (propofol)
    - Regional anesthesia/analgesia – intraoperative and postoperative
    - Hydration
    - Nonopioid analgesics (nonsteroidal anti-inflammatory agents)
  - Use ≥2 antiemetics of different classes prophylactically (eg, dexamethasone + ondansetron[a])
    - Serotonin antagonists (ondansetron[a], granisetron, dolasetron; ondansetron[a] is usual first-line drug)
    - Steroids (dexamethasone)
    - Antihistamine (dimenhydrinate, cyclizine)
    - Antidopaminergic (droperidol[b])
    - Phenothiazine (perphenazine)
    - Neurokinin-1 antagonist (aprepitant)
  - Treat postoperative nausea and vomiting
    - Serotonin antagonist or antiemetic drug of different class

[a]Ondansetron is approved in United States for postoperative vomiting in children ages 1 month and older.
[b]In the United States, droperidol has a black box warning as it causes a dose-dependent prolongation of the QT interval.

Preoperative fasting does not prevent PONV, but hydration and glucose supplementation will ameliorate it. Serotonin antagonists are the mainstay of pharmacologic management of POV/PONV due to their safety profile and minimal side effects. Ondansetron (0.15 mg/kg IV every 4-6 hours) is the most common first-line drug for POV/PONV prophylaxis and treatment, and it is the only serotonin antagonist that is approved for use in children ages 1 month and older in the United States. The combination of ondansetron and dexamethasone (0.5 mg/kg IV) is efficacious when used prophylactically.

IX. NUTRITIONAL CONSIDERATIONS

Acute malnutrition is frequently encountered in the postoperative pediatric patient. In this setting, malnutrition is multifactorial and may be caused by prolonged bowel dysfunction or food intolerance after surgery, increased catabolism, lack of aggressive intervention, or missed opportunities to increase nutritional support. To complicate the picture further, many children have long-term malnutrition due to a preexisting chronic condition.

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The unique challenge in the pediatric patient is providing nutrition to meet the increased demands of critical illness and healing, as well as the demands of normal growth and development.

A. Nutrition Assessment

On admission, all patients should be assessed for evidence of malnutrition. Baseline measures, such as weight, height, and head circumference, should be obtained. Children younger than 2 years should be plotted on normative growth charts, and values can be compared with previous measures, if available. Body mass index can be determined in older children. Also, base measures of specific visceral proteins, such as albumin, transferrin, prealbumin, and retinol-binding protein, can be obtained. These proteins have a rapid turnover, but during periods of increased catabolism, their synthesis is decreased. Increasing levels are indicative of resolving stress and/or adequate nutrition. Prealbumin and retinol-binding protein, due to their short half-lives (2 days and 12 hours, respectively), are better suited to reflect acute change in the patient's metabolic state. Another readily available biochemical marker is C-reactive protein; in postsurgical patients, a normalizing level heralds a returning anabolic state.

B. Stress Response

During acute physiologic stress, regardless of the inciting event, the body's response tends to be the same. The hypothalamic-pituitary axis and the sympathetic nervous system are activated, resulting in an increase in counter-regulatory hormones, including endogenous catecholamines and eventually insulin resistance. Elevated cortisol induces muscle breakdown and promotes gluconeogenesis (conversion of noncarbohydrate energy sources into glucose). The increased levels of catecholamines stimulate glycogen breakdown in the liver and skeletal muscle and mobilization of free fatty acids, all of which fuel the hypercatabolic state.

C. Nutritional Support

The breakdown of lean body tissue is central during the stress response. Therefore, the goal is to limit protein breakdown during the acute phase of illness by providing amino acids and adequate nonprotein calories (**Table 19-6**).

Table 19-6 NUTRITIONAL REQUIREMENTs

Age (years)	Energy (kcal/kg/day)	Protein (g/kg/day)
<1	50-80	2.0-2.5
1-7	45-65	1.5-2.0
>7	30-60	1.5-2.0

(*1 kcal = 4.2 kJ)

Carbohydrates in the form of glucose provide 4 kcal/g of energy. Children require a glucose infusion

rate of 5 to 8 mg/kg/min to meet metabolic demand. The threshold for ketosis is a glucose infusion rate of less than 2 mg/kg/min, but this may be higher in the context of the stress response due to the state of insulin resistance. Overfeeding with glucose delivery rates of more than 10 mg/kg/min should be avoided, because such rates promote lipogenesis and increased CO_2 production. If hyperglycemia develops despite appropriate glucose delivery, insulin supplementation (starting continuous infusion of regular insulin, 0.05-0.1 U/kg/h) in a monitored environment may be indicated to facilitate cellular uptake. When reintroducing enteral nutrition, a lactose-free formulation may be tolerated best.

Fats provide 9 kcal/g of energy. In patients unable to tolerate enteral feedings, fat can be administered parenterally as a 20% emulsion. As little as 0.5 g/kg/d is enough to provide essential fatty acids (linoleic and linolenic acids), but the infusion can be increased by 1 g/kg/d to a limit of 3 g/kg/d, depending upon caloric requirements. Triglyceride levels are best determined at least 4 hours after discontinuing a lipid infusion. Hypertriglyceridemia is frequently caused by an excess caloric load from carbohydrates. When the enteral route is used, the medium-chain triglycerides offered in some formulas are able to enter the portal system directly, facilitating absorption.

The breakdown of protein produces 4 kcal/g of energy. Although amino acids are unable to prevent the catabolism of proteins during the stress response, they should be provided in an amount sufficient to prevent a deficiency and to facilitate the synthesis of new protein. Maintaining this balance between breakdown and synthesis may require protein intake of 2 to 3 g/kg/d.

D. Route of Administration

The use of IV delivery devices and central venous catheterization allows the administration of parenteral nutrition to surgical patients who would otherwise suffer the consequences of malnutrition (eg, poor wound healing, infections, even death).

Enteral nutrition is the preferred mode of nutrient intake in critically ill patients with a functional gastrointestinal system, because of its many benefits, lower cost, and lower complication rate compared to parenteral nutrition (**Table 19-7**).

Table 19-7	ADVANTAGES OF ENTERAL FEEDING

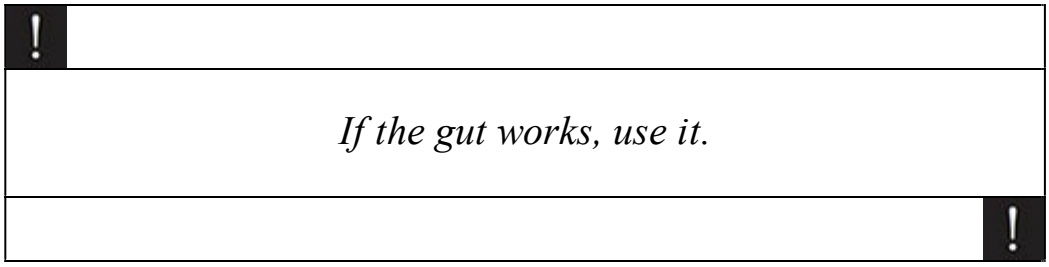
- Stimulates release of gut trophic factors
- Maintains enterocyte mass
- Preserves gut motility
- Improves mesenteric blood flow
- Decreases incidence of nosocomial infections
- Lowers costs

Delayed gastric emptying is common in critical illness. If attempts to feed into the stomach fail or if the risks of aspiration are high, transpyloric tube feeding is usually successful and has the added advantage that feeding can be continued around any planned extubation and during surgery or

procedures, providing uninterrupted nutrition. Parenteral nutrition continues to play an important role in the support of patients with hemodynamic instability at risk for bowel ischemia and in patients with congenital or acquired abnormalities of the gut, such as necrotizing enterocolitis, bowel atresia, meconium ileus, dysmotility syndromes, bowel obstruction, and short gut syndrome. Complications of prolonged parenteral nutrition are numerous, and every effort should be made to provide some nutrition by the enteral route (**Table 19-8**).

Table 19-8	COMPLICATIONS OF PARENTERAL NUTRITION

- Catheter-related infections
- Electrolyte disturbances
- Hepatic disorders
  - –Fatty liver
  - –Cholestasis
  - –Hepatic fibrosis
  - –Biliary cirrhosis (parenteral nutrition-associated liver disease)



X. HEMATOLOGIC CONSIDERATIONS

Intraoperative blood loss should be carefully monitored by both the surgeon and the anesthesiologist. Large volumes of blood products given intraoperatively and ongoing bleeding can have a major impact on the patient's status in the postoperative period. When the blood loss has been gradual, the rates at which the hemoglobin and hematocrit fall can be used as an estimation of the extent of the loss. When active bleeding is vigorous, hemodynamic instability is a closer reflection of blood volume depletion. Direct measurement of blood collected from indwelling drains (eg, thoracostomy tubes) is also used to estimate the extent of ongoing blood loss. Occult bleeding occurring in closed compartments, such as the intra-abdominal compartment and thigh, is an exception to this practice. The etiology of postoperative bleeding must be determined and corrected, especially if it is ongoing or brisk. The most common cause for bleeding is poor surgical hemostasis. Other etiologies are coagulopathy due to hemodilution of clotting factors and platelets, increased consumption, massive packed red blood cell (PRBC) transfusion, or other underlying conditions, including sepsis and disseminated intravascular coagulopathy.

The decision to transfuse depends on the patient's clinical status and the estimated rate of blood loss.

The clinical evidence does not support transfusion of PRBCs to hemodynamically stable patients unless the hemoglobin is <7 g/dL. Massive blood loss is defined as the loss of 1 blood volume within a 24-hour period or the loss of 50% of blood volume within 3 hours. Estimated blood volume is 85 mL/kg in infants (0-1 year), 80 mL/kg in children (1-10 years), between 70 and 75 mL/kg in adolescents, and 65 mL/kg in adults. In the event of ongoing massive bleeding, the patient initially should be resuscitated with PRBCs (10-20 mL/kg) via large-bore IV access instead of with colloids or crystalloids. Simultaneously, baseline studies should be obtained: complete blood count, coagulation screening, and blood typing and screening. Blood product replacement should be guided by the estimated volume loss, rate of ongoing loss, and the patient's response to fluid resuscitation (**Chapter 17**). The therapeutic goals are maintaining blood volume or hemodynamic parameters and achieving a hemoglobin level >7 g/dL. Fresh frozen plasma and platelets should be used judiciously to prevent or correct any coagulopathy. To address coagulopathy early in massive bleeding, near-equal amounts of fresh frozen plasma and PRBCs (1:1 ratio) should be transfused. The need for platelets is variable. A platelet count >20,000/μL may be acceptable in the absence of ongoing bleeding, but a count >50,000/μL is desirable in active bleeding. Patients who have undergone a neurosurgical procedure should maintain platelet counts ≥100,000/μL. In infants and children, a platelet transfusion of 10 mL/kg will increase the platelet count by approximately 50,000/μL. The priority, however, is to surgically reverse the source of the bleeding.

PRBCs and fresh frozen plasma contain citrate, which chelates calcium and can cause a fall in the serum ionized calcium level. The mobilization of calcium and the hepatic metabolism of citrate are usually rapid enough to prevent a significant fall in the ionized calcium in older children and adults. Neonates have inadequate calcium stores and are susceptible to acute hypocalcemia and its attendant hypotension, so they require IV calcium replacement (calcium chloride 10%, 10-20 mg/kg, or calcium gluconate, 100-200 mg/kg). Calcium may also be required if massive amounts of blood products have been transfused. Large transfusions of cold blood products will precipitate hypothermia, so the products should be warmed prior to administration.

XI. THERMAL REGULATION

During intraoperative management, the anesthesiologist optimizes body temperature control, with the primary goal being normothermia. Some patients are prone to develop hypothermia or hyperthermia during anesthesia. Hypothermia is a major concern in infants, in patients having prolonged procedures, after the administration of large volumes of unwarmed IV fluids or blood products, and in instances that require a large surgical site exposure. Hyperthermia is possible when radiant warmers or thermal blankets are used.

Core temperature measurements from the tympanic membrane, distal esophagus, nasopharynx, or pulmonary artery are reliable. Skin temperature varies with the environmental temperature. Rectal and bladder temperature do not follow any rapid change in body temperature.

Infants and small children are at increased risk for

hypothermia because of their ratios of body surface area to mass, high metabolic rates, and low fat stores.

A. Hypothermia

Shivering is common in the immediate postoperative period with patients often complaining of feeling extremely cold. The use of warm blankets may remedy this and tends to be comforting; subjectively, it appears to reduce shivering. Hypothermia (core temperature <36°C [96.8°F]), on the other hand, can be a cause of shivering and must be ruled out. If not promptly attended to, hypothermia can lead to life-threatening situations, especially in neonates who use non-shivering thermogenesis (**Table 19-9**). During rewarming, cutaneous hyperthermia and burns (especially with improper use of radiant warmers) must be avoided.

Table 19-9 DELETERIOUS EFFECTS OF HYPOTHERMIA

- Respiratory acidosis
- Apnea
- Hypotension
- Bradycardia
- Prolongation of neuromuscular blockade
- Prolongation of the effects of narcotics

B. Fever

Postoperative fever (temperature >38°C [>100.4°F]) is common, managed expectantly with symptomatic treatment, and usually resolves spontaneously. The onset of postoperative fever is important in establishing a differential diagnosis and will determine the workup and management. Fever in the first 24 hours postoperatively is relatively common and is often due to the inflammatory response from surgery-induced tissue injury, resulting in the release of inflammatory mediators. The patient may have had a preexisting infection at the time of surgery. Healthcare-associated infections are the most common causes of fever. These include hospital-acquired pneumonia, catheter-associated urinary tract infection, central line-associated bloodstream infection, and surgical site infection. Pneumonia usually presents on postoperative day 2 or 3, with urinary tract infections appearing on days 3 through 5 and surgical infections on days 5 through 7.

Interventions that can reduce healthcare-associated infections in postoperative patients (**Table 19-10**) include extubating mechanically ventilated patients and removing indwelling catheters as soon as possible. Appropriate cultures should be obtained, and antibiotics should be instituted as needed

(Chapter 7). Persistent postoperative fever may not be caused by an infectious agent, and other causes should be sought. Using the process of elimination, the underlying etiology can often be determined and treated.

Table 19-10 REDUCING THE RISK OF POSTSURGICAL HEALTHCARE-ASSOCIATED INFECTIONS

- Hospital-acquired pneumonia
  - –Minimize ventilator days
  - –Keep head of bed elevated 30°
  - –Ambulate out of bed quickly when age and developmentally appropriate
  - –Encourage or improve deep breathing in nonventilated patient
    - –Incentive spirometry (4-6 years or older)
    - –Blowing bubbles or pinwheel (toddler or older)
- Hospital-acquired infections
  - –Minimize use and duration of indwelling catheters and lines
- Proper hand hygiene
- Surgical site infections
  - –Administer prophylactic antibiotic 1 h before incision
  - –Discontinue prophylactic antibiotics within 24 h after surgery end time (48 h in cardiac patients)
  - –Remove hair as appropriate (leave hair intact or remove with clippers)

Another cause of hyperthermia is excessive ambient environmental heat with no opportunity for heat loss, as can be seen with infant bundling and the use of warming devices. Dehydration secondary to inadequate fluid replacement also predisposes the patient to temperature elevation. In all cases, fever results in increased metabolic demand with resultant tachycardia, tachypnea, and carbon dioxide production, causing increased stress. Malignant hyperthermia should be considered in patients who have a temperature in excess of 39°C (102.2°F) during or within hours of receiving an inhalational anesthetic.

C. Malignant Hyperthermia

Malignant hyperthermia (MH) is a familial disorder of muscle hypermetabolism that may complicate the anesthetic course and require further management in the pediatric ICU. Its prevalence during general anesthesia varies: 1 in 15,000 children, 1 in 50,000 adults. Children with underlying myopathy are at increased risk. Agents aggravating MH include halogenated inhalational anesthetic agents and depolarizing muscle relaxants, such as succinylcholine. Exaggerated jaw rigidity (masseter spasms) after succinylcholine use and excessive carbon dioxide production are often the earliest signs; skeletal muscle rigidity, tachycardia, and hyperthermia then ensue. If not controlled, the intense muscle activity leads to rhabdomyolysis, hyperkalemia, metabolic and respiratory acidosis, and eventual multiple organ failure. Immediate evaluation for MH should include measurement of arterial blood gases, serum electrolytes, serum creatinine kinase, and urine myoglobin. If MH is suspected, an

anesthesia or pediatric intensivist consult should be obtained urgently to assist in diagnosis and medical management. The diagnostic triad consists of skeletal muscle rigidity, metabolic acidosis, and elevated body temperature.

Treatment consists of avoidance or immediate removal of triggering agents, muscle relaxation with dantrolene sodium, and supportive care. Dantrolene sodium causes complete and sustained relaxation of skeletal muscles by direct action on calcium release channels. It is given in a dose of 1 to 3 mg/kg IV, repeated every 15 minutes as needed, to a maximum of 10 mg/kg in the setting of acute MH. Recurrence is prevented by administration of dantrolene sodium, 1 mg/kg IV every 6 hours for 24 to 48 hours postoperatively. Supportive care involves airway protection with orotracheal intubation and mechanical ventilation if large doses are needed, active cooling to <38ºC (<100.4ºF) but avoidance of hypothermia, and correction of acidosis and hyperkalemia. Laboratory testing should include creatine kinase, phosphate, calcium, and myoglobin. In the presence of rhabdomyolysis and myoglobinuria, a brisk urine output should be maintained with IV hydration and osmotic diuretics, such as mannitol. As with other hyperthermia syndromes, coagulopathy, pulmonary edema, and cerebral edema can be potential complications. When MH is suspected based on well-documented clinical events or strong family history, genetic evaluation and muscle testing could secure a diagnosis and indicate appropriate precautions for both the index patient and relatives of all ages.

Key Points

Postoperative Management

- Postoperative recovery is directly dependent on preexisting conditions, severity of illness, and the duration of anesthesia.
- Adverse respiratory events are the most common complication arising within the first 24 hours after surgery.
- Full-term neonates (younger than 1 month) and premature infants less than 60 weeks' postconceptual age should be considered at risk for apnea after general anesthesia, and they require inpatient monitoring and observation for 24 hours after surgery.
- Naloxone should be used if respiratory or central nervous system depression is suspected because of excessive opioid use; however, it should be used judiciously, as it not only reverses the depressive effects but also the analgesia, resulting in a child who is in pain, agitated, hypertensive, and behaviorally uncontrollable.
- Emergence agitation is usually treated with opioids, benzodiazepines, and hypnotics, but other causes of agitation – such as hypoxia, hypoglycemia, and severe pain – must be ruled out.
- Children of all ages feel pain and must be aggressively managed in the postoperative period. A developmentally appropriate pain scale is a vital tool in the assessment of the pain intensity.
- A balanced and multimodal approach to pain management, tailored to the patient, delivers

effective analgesia and minimizes adverse drug events.

- Volume deficit, the most common fluid problem in the pediatric surgical patient, is usually due to third spacing and ongoing loss of gastrointestinal fluids, but other causes of hemodynamic lability must be ruled out
- In the critically ill postoperative patient, it is safe to assume that any ongoing fluid losses are likely to be isotonic in composition.
- Multimodal therapy should be used to prevent and treat PONV in children.
- When active bleeding is vigorous, hemodynamic instability is a closer reflection of blood volume depletion than laboratory results.
- Early institution of nutrition, preferably by the enteral route, shortens the postoperative period and speeds recovery.
- Fever is common after surgery and its onset time is important in establishing a differential diagnosis and determining the workup and management.
- A patient with the diagnostic triad of skeletal muscle rigidity, metabolic acidosis, and elevated body temperature within hours of receiving an inhalational anesthetic has malignant hyperthermia until proven otherwise.

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SEDATION, ANALGESIA, AND NEUROMUSCULAR BLOCKADE

Objectives

- Identify the indications, risks, and monitoring needs associated with patients undergoing elective sedation or intubation.
- Describe the benefits and appropriate uses of therapeutic analgesia in critically ill patients.
- Explain the benefits and side effects associated with commonly used analgesic medications.
- Outline the benefits and side effects associated with commonly used sedative/hypnotic medications.
- Discuss the benefits and side effects associated with commonly used neuromuscular blockade medications.
- Identify the reversal medications for benzodiazepine, opioid, and neuromuscular blocking agents.
- Recognize the complications of tachyphylaxis and iatrogenic dependence associated with the prolonged usage of benzodiazepine and opioid receptor agonists.

Case Study

A 10-year-old boy presents to the emergency department after falling from his bike. He is alert and in obvious pain. He has a facial laceration to the right maxillary area and an obvious deformity of his right forearm with a deep laceration overlying it. His vital signs are as follows: heart rate, 104 beats/min; respiratory rate, 27 breaths/min; blood pressure, 103/71 mm Hg; oxygen saturation by pulse oximetry, 99% on room air. Radiographs demonstrate a displaced radio-ulnar open fracture. The orthopedic and plastic surgery consultants request procedural sedation to facilitate reduction of the fracture, exploration of the wound, and repair of the facial laceration. The patient has a history of moderate persistent asthma. His last meal was 5 hours ago.

Detection

– Is this patient a good candidate for procedural sedation?

Intervention

– What are the most immediate treatment strategies?

Reassessment

– What medications can be given to provide analgesia and sedation for his necessary procedures?

– Which medicines should be avoided and why?

– What complications are the most likely in the performance of procedural sedation?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will this information be disseminated?

– Where is the best place to manage the care of this patient?

Teamwork

– How should this treatment strategy be implemented?

– Who is to do what and when?

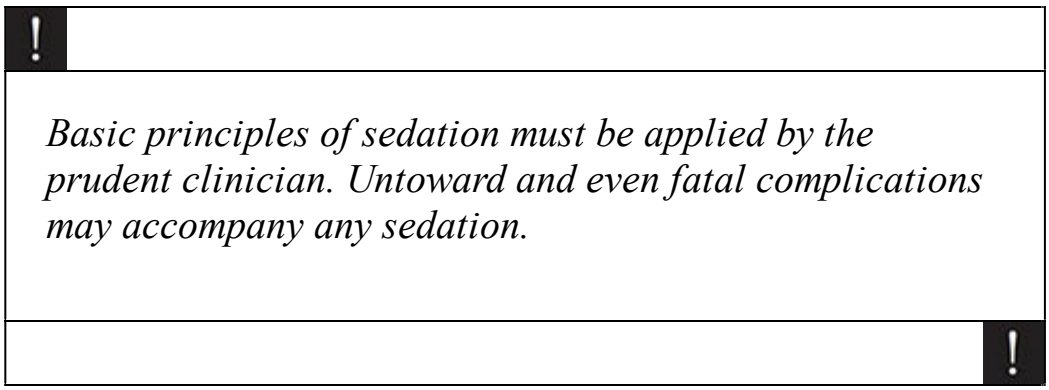
I. INTRODUCTION

The treatment of critically ill children would be difficult, if not impossible, without the concurrent use of analgesic, sedative, and neuromuscular blocking agents. When used properly, they allow painful and traumatic procedures to be performed on severely ill children. These procedures include the placement of artificial airways, invasive monitoring devices, central access devices, and even minor surgery. Long-term analgesia and sedation may be required after insertion to maintain these treatments and protect the patient's safety and welfare until these devices are no longer needed. Sedative medications may also be needed for patients undergoing procedures that are not painful, such as computed tomography scans or magnetic resonance imaging, especially for infants or toddlers who have a difficult time remaining still for prolonged periods.

By definition, a patient who receives a medication to decrease stress or anxiety is receiving sedation. Anyone receiving a medication or therapy to relieve pain is receiving analgesia. Some medications provide sedation without analgesia, whereas others provide analgesia without sedation; a few provide both. Each practitioner should determine which of these conditions they want to provide and then choose the appropriate medication, or combination of medications, to accomplish this therapeutic goal.

Sedation in the critical care environment reduces metabolic demands, mortality, morbidity, and hospital costs. More importantly, the treatment of critical illness may not only be painful, but may be frightening as well. In fact, the care of critically ill children can lead to long-term psychological

trauma and stress. Use of appropriate sedation and analgesia can reduce the negative impact of these factors. However, each medication has its own indications and side effects, which must be weighed in terms of benefits and risks for each patient on a case-by-case basis. Used correctly, these medications have incredible benefits. Used incorrectly, or without an appropriate level of caution, they can cause significant harm and even death.


!
Basic principles of sedation must be applied by the prudent clinician. Untoward and even fatal complications may accompany any sedation.
!

II. PLANNING FOR SEDATION

Each medication has its intrinsic benefits. Determining the correct medication to use, or avoid, requires careful planning, based on the particular situation, to prevent significant harm or complications. The overriding goal of any sedation plan should be the use of the least analgesia or sedation needed to accomplish the therapeutic goal. All patients are different, and their responses to any medications can vary. These consequences can be reduced or eliminated by careful assessment and preplanning.

Several questions must be asked before beginning any sedation:

- What is the goal of this sedation?
- What length of sedation is required?
- What complications are possible? Likely?

First, the most important question: What is the goal of this particular sedation? If only analgesia is needed, the practitioner must determine how long an effect will be required. If the goal is simply to complete a short painful procedure, perhaps the best choice is a short-acting analgesic with minimal or no sedative component. Alternatively, if the procedure requires a prolonged period and is particularly painful or traumatic, perhaps a better choice is a combination of analgesic and sedative medication.

What complications are possible? Is the patient at risk of losing the airway during the procedure? If the risk is high, long-acting medications that depress respiration should be avoided, and the practitioner should consider elective intubation before the procedure. If the risk of airway loss is low, a different choice of medications may be made. Is the patient hemodynamically unstable? A radically different choice of medications may be required compared to those used in a relatively stable patient. It is recommended that only anesthesiologists or physicians with specialized training

perform sedation in hemodynamically unstable patients.

Lastly, providers must question themselves and their own skills. What if this child should lose airway protective reflexes and require an invasive airway? Should a more experienced provider be brought in to help before beginning the sedation? Similarly, how emergent is the procedure? Should an anesthesiologist be performing the sedation? All of these questions must be addressed before any medications are given in order to protect the patient's life and to prevent unnecessary morbidity.

A. American Society of Anesthesiologists Classification System

The American Society of Anesthesiologists (ASA) Physical Status Classification System (**Table 20-1**) was developed to classify the severity of illness in patients about to undergo anesthesia. The system has 6 physical status levels, ranging from a healthy individual (ASA PI) to patients who are brain dead (ASA PVI). Although the system is not meant to predict outcomes, it does provide a general assessment of preoperative risk. Most importantly, those who are not anesthesiologists should be cautious or even avoid electively sedating patients of ASA PIII level and above. Similarly, they should always avoid electively sedating patients of ASA PIV level and above if at all possible.

Table 20-1	AMERICAN SOCIETY OF ANESTHESIOLOGISTS PHYSICAL STATUS CLASSIFICATION SYSTEM
ASA PI:	Normal healthy patient
ASA PII:	Mild systemic disease, with no functional limitations
ASA PIII:	Severe systemic disease which limits activity but is not incapacitating
ASA PIV[a]:	Incapacitating disease that is a constant threat to life
ASA PV[a]:	Patients not expected to survive 24 hours with or without an operation
ASA PVI:	Brain-dead patient being prepared for organ donation

[a]It is recommended that these patients receive sedation administered by appropriately trained anesthesiologists and intensivists.

Reproduced from American Society of Anesthesiologists guideline on physical status classification system. http://www.asahq.org/quality-and-practice-management/standards-and-guidelines-and-related-resources-search. Published October 15, 2014. Accessed May 28, 2018.

B. Depth of Sedation

Current classification divides sedation into four levels (**Table 20-2**). The levels are defined by four different parameters corresponding to the presumed "depth" of sedation: responsiveness, airway maintenance, spontaneous respiration, and cardiovascular function. The level of sedation, and the corresponding patient response, is a continuum without clear delineation of transition from one level to the next. Likewise, a weight-based drug dosage that provides minimal sedation in one patient may cause general anesthesia in another. Similarly, a weight-based dosage that provided minimal sedation

to a healthy patient may cause general anesthesia or cardiovascular collapse in the same patient in the face of critical illness, renal dysfunction, or hepatic failure. For these reasons, providers should attempt to provide the minimum level of sedation required to accomplish the therapeutic goal in each patient. The policy of "start low and go slow" (ie, use low doses and slowly repeat or increase doses as needed) allows the practitioner to titrate to the required, but minimum, level of sedation. While trying to achieve the minimal level, it is also important to use therapeutic dosages. A common mistake is to use sub-therapeutic doses, which results in inadequate sedation that still exposes the patient to potential side effects. A patient can always drift into a deeper-than-intended level of sedation despite best efforts, and the practitioner must always be prepared to actively manage the airway, maintain oxygenation and ventilation, and support cardiovascular function, as indicated.

Table 20-2 LEVELS OF SEDATION

Levels of Sedation	Response	Cardiopulmonary Effects
Minimal sedation	Normal response to verbal stimulation	Ventilation/airway reflexes intact No cardiovascular effect
Moderate sedation/analgesia (formerly conscious sedation)	Responds purposely to verbal commands or to light tactile stimulation	Maintains patient airway, ventilation adequate Minimal cardiovascular effects
Deep sedation	Not easily aroused, but responds purposely to repeated or painful stimuli	May require assistance to maintain airway and/or ventilation Cardiovascular function usually maintained
General anesthesia	Unconsciousness, unresponsive to physical or verbal commands	Loss of airway reflexes, requires assistance to support ventilation Cardiovascular function may be impaired

Reproduced from American Society of Anesthesiologists guideline on continuum of depth of sedation: definition of anesthesia and levels of sedation/analgesia. http://www.asahq.org/quality-and-practice-management/standards-and-guidelines-and-related-resources-search. Published October 15, 2014. Accessed May 28, 2018.

C. Risks of Encountering a Difficult Airway

Anyone performing sedation must be prepared in case the patient loses airway protective reflexes at any time, regardless of the targeted level of sedation. Therefore, the practitioner must always have a plan for advanced airway management in any patient, as well as the ability and training to execute that plan. For this reason, the provider must attempt to predict how likely any patient is to have a difficult airway prior to administration of any medication. A difficult airway, by definition, is challenging or impossible to maintain by routine or straightforward measures. Occurrence of difficult airway in pediatric patients is reported to be between 0.9% and 2.1%. Any patient, regardless of history, may have a difficult airway, resulting in an increased number of attempts at airway insertion or difficulties with mask ventilation or oxygenation. Difficulty cannot be definitively determined until advanced airway management is attempted, but several factors can suggest a high, or at least increased, risk of encountering a difficult airway (**Table 20-3**).

Table 20-3	CLINICAL CONDITIONS AND SYNDROMES ASSOCIATED WITH DIFFICULT AIRWAY[a]

- History of difficult intubation
- History of complications with anesthesia
- Family history of difficulty with intubation or anesthesia
- History of obstructive sleep apnea, snoring, tracheomalacia, or laryngomalacia
- Pharyngeal and laryngeal pathology
- Bronchospasm
- Stridor at rest
- Chromosomal abnormalities (trisomy 13, 18, or 21)
- Pierre Robin sequence
- Beckwith-Wiedemann syndrome
- Treacher Collins syndrome
- Micrognathia or retrognathia
- Obesity
- Short neck
- Poor neck extension or fused cervical vertebrae
- High arched palate
- Macroglossia
- Hemifacial microsomia
- Small thyromental or hyomental distance
- Tracheal deviation
- Facial or cervical trauma, mass, or infection
- Trismus
- Mallampati II classification or greater

[a]Bag-valve mask ventilation and/or intubation

Data from American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Practice guidelines for sedation and analgesia by non-anesthesiologists. *Anesthesiology*. 2002; 96(4):1004-17.

The Mallampati classification (**Figure 20-1**) is a simple airway assessment technique that involves looking in the patient's open mouth with and without phonation. The Cormack-Lehane airway classification (**Figure 20-2**) grades the appearance of the glottic opening during laryngoscopy. In both classification systems, higher grades correspond with the potential for difficult airway. If any features suggestive of a difficult airway are present by either examination or by history (**Table 20-3** and **Figures 20-1, 20-2**), the provider should consider involving an anesthesiologist before sedating or intubating a patient, especially for an elective procedure. In addition, the provider should be familiar with the use of the difficult airway algorithm (**Appendix 8**), regardless of the presence or absence of risk factors. It is far better to plan for a controlled intubation in a patient with a potentially difficult airway than to emergently intubate during a procedure.

Figure 20-1. Mallampati Classification

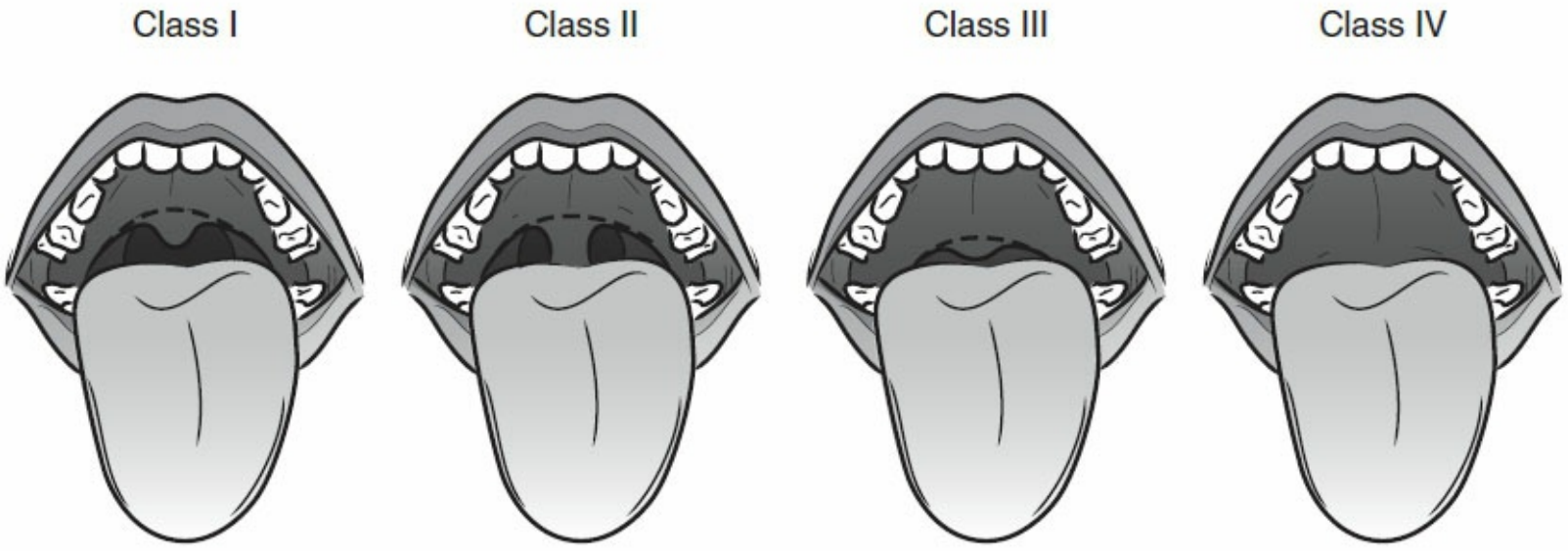
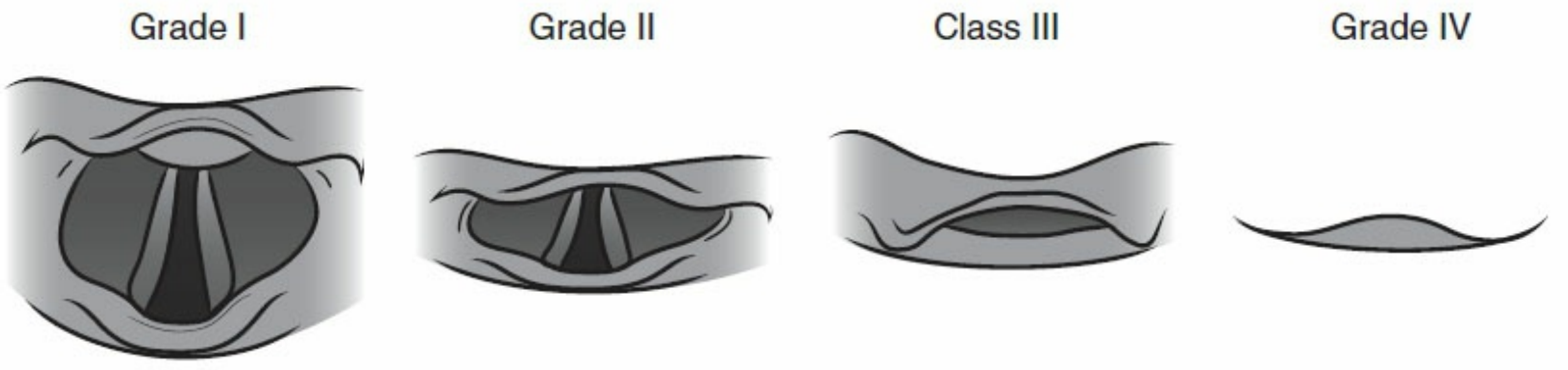


Figure 20-2. Cormack-Lehane Airway Classifications



D. Patient History

A thorough history should be taken prior to every sedation event to decrease the risk of avoidable complications.

- Does the patient have any history of difficulty with anesthesia? This may include sensitivity to particular drugs, difficulty with obtaining an airway, prolonged effects of anesthesia medications, or any other significant history that may impact safety.
- Is there a history of difficulty with anesthesia or sedation in the parents or other family members? This may uncover familial conditions that differentially affect the metabolism of medications, especially entities such as malignant hyperthermia or acetylcholinesterase deficiency.
- Is the patient allergic to any drugs that may be given during sedation procedures? Without knowing the history, those medications cannot be avoided.
- Are there other known allergies?
- Have there been any recent illnesses? Especially in elective cases, concurrent upper airway infections can cause difficulties with airway patency and adequacy of ventilation. A recent illness may indicate that sedation should not be performed, if possible, until the patient is recovered.
- Does the patient have known renal or hepatic dysfunction? Several medications used in analgesia and sedation utilize liver or kidney metabolism and must be avoided, or have their dosages reduced, in patients with dysfunction.
- Have all available laboratory results been reviewed? Finding a low platelet count or severe coagulopathy after attempted placement of an invasive line can be disastrous.
- Have all imaging studies been reviewed? If the patient is symptomatic because of a large intracranial mass, many drugs should be avoided. Likewise, the presence of a chest mass dictates that even minimal sedation is contraindicated by anyone except a qualified anesthesiologist.

- How long has the patient been fasting? The ASA has published fasting guidelines (**Table 20-4**). Vomiting during sedation can turn a routine procedure into a life-threatening event. ASA guidelines apply to elective sedations only. In some situations, emergent sedation may be needed. When the guidelines cannot be followed because of emergent needs, the provider must balance the increased risk of vomiting and aspiration against the benefit of sedation.

Table 20-4 AMERICAN SOCIETY OF ANESTHESIOLOGISTS GUIDELINES FOR PREOPERATIVE FASTING

Substance Ingested	Minimum time to sedation following ingestion
Clear fluids	2 hours
Breast milk	4 hours
Infant formula/nonhuman milk	6 hours
Light meal (toast, cereals)	6 hours
Heavy meal (fatty foods, meats)	8 hours

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III. PRE-SEDATION SETUP AND MONITORING

A. Setup

Once a sedation goal has been determined and all risks that can be easily discovered have been identified, a successful sedation requires extensive setup before any medication is administered. The ASA has defined the minimum equipment and monitoring required for sedation provided by clinicians who are not anesthesiologists (**Table 20-5**). Cardiorespiratory monitoring and continuous pulse oximetry should always be used. The growing availability of end-tidal CO_2 monitoring, preferably as capnography, makes it an indispensable tool in the care of the sedated and/or intubated patient. Trends in end-tidal CO_2 and changes in waveform can signal complications and allow for interventions long before oxygen desaturation occurs. If all required equipment is not available and ready for use, the sedation induction should not be initiated.

Table 20-5 MINIMAL EQUIPMENT AND MONITORING NECESSARY FOR SEDATION

Cardiac monitor
- 3- to 5-lead electrocardiography

Pulse oximetry
Respiratory or apnea monitor
End-tidal CO_2 monitor
Working access (ie, peripheral intravenous or central venous line), preferably 2
Resuscitation fluid running (normal saline or lactated Ringer solution)
Oral pharyngeal airway
Nasogastric tube
Bag-mask ventilation

Endotracheal tube
- Correct size
- 1 size larger and 1 size smaller

Drugs appropriate for sedation drawn and labeled
Suction with Yankauer tip, on and running
End-tidal CO_2 detector for endotracheal tube placement
Laryngeal mask airway or other backup airway device
- Oral pharyngeal airway
- Nasopharyngeal airway

• Appropriate size • Oxygen flowing	Laryngoscope blade • Appropriate size for patient • Macintosh or Miller blades • Stylet, size appropriate for endotracheal tube

Reproduced with permission. © 2002 Wolters Kluwer Health. American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists. Practice guidelines for sedation and analgesia by non-anesthesiologists. *Anesthesiology*. 2002;96:1004-1017.

A useful mnemonic for airway equipment setup in all patients is ON BED SCABS:

Oral pharyngeal airway

Nasogastric tube

Bag and mask appropriate for this child, with oxygen flowing

Endotracheal tube of appropriate size, 1 size larger and 1 size smaller

Drugs appropriate for sedation and reversal, drawn and labeled

Suction on and running (with Yankauer tip attached) and **S**taff

CCO_2 detector for endotracheal tube placement

Alternate airway and alternate plan (ie, laryngeal mask airway)

Blade (laryngoscope) appropriate for this patient, with light working

Stylet appropriate for endotracheal tube choice

A more familiar mnemonic is SOAPME:

Suction and **S**taff

Oxygen (2 sources)

Advanced airway equipment

Pharmacy to provide emergency medications and antagonists

Monitoring appropriate for the level of sedation

Equipment for emergency resuscitation (eg, code cart with defibrillator)

All equipment should be set up for use at the bedside and checked before sedation is initiated. "Nearby" is not close enough and can lead to complications. Resuscitation crystalloids should be prepared and ready; this can be achieved either by having 20 mL/kg boluses predrawn in syringes, or via an inline system using a 3-way stopcock and a 60-mL syringe to perform "pull-push" infusions. No fluids containing potassium should be in the room as they may be inadvertently used during resuscitation. Lastly, the person monitoring sedation should never be the person performing the procedure. Failure to follow this recommendation can lead to a critical delay in recognizing a change

in vital signs requiring immediate intervention. Although this setup may seem excessive, thorough preparedness can be the difference between life and death in an emergency.

B. Critical Communications and Team Organization

A time-out should be called prior to any procedure, as required by the Joint Commission for all invasive procedures. It is a standardized process to improve patient safety and safeguard against wrong patient, wrong procedure, wrong site errors. At minimum it must include verification of patient identity, site for procedure, and procedure to be performed. Additionally, any pertinent imaging studies should be immediately available. Many institutions utilize the time-out for additional procedures. Procedural sedation is considered a procedure in its own right and requires a separate time-out.

Another process that can improve patient safety is the pre-procedural huddle. This is led by the provider performing the procedure and includes:

- Identification of each person present and the role each plays
- Specifics of the procedure; for sedation, this includes medications that will be used and specific doses, as well as verification that the medications have been prepared
- Any medical concerns regarding the patient
- Verification that the equipment needed is present and ready for use

During or at the end of the huddle, team members are given an opportunity to ask questions or offer any additional information.

IV. DRUG ADMINISTRATION DURING SEDATION OR INTUBATION

Medication administration during all sedation and intubation procedures should use a rational and consistent methodology including pre-oxygenation, premedication, sedation, and relaxation.

Case Study

A 14-month-old female child is brought to the emergency department with severe respiratory distress. Vital signs are as follows: temperature, 38.6°C (101.4°F); heart rate, 157 beats/min; respiratory rate, 47 breaths/min; blood pressure, 44/28 mm Hg; oxygen saturation on pulse oximetry 82% on room air, 88% on non-rebreather mask. Her weight is 9.5 kg (21 lbs). Her medical history is significant for premature birth at 28 weeks' gestation. She had a prolonged neonatal ICU course with a 3-week intubation and has residual subglottic stenosis. She sustained a grade III intraventricular hemorrhage and has pronounced cerebral palsy with motor and mental developmental delays. This child has

severe dysphagia and failure to thrive, and she is dependent upon gastric tube feedings. Her mother reports emesis following a feeding this morning and, since that time, the girl has had worsening breathing difficulties.

Detection

– What is the likely diagnosis in this patient?

– What other considerations should be included in the differential diagnoses?

Intervention

– What are the most immediate treatment strategies?

– What findings would prompt intubation?

Reassessment

– What medications can be given to provide analgesia and sedation for the necessary procedures?

– Which medicines should be avoided and why?

– What special considerations affect the management of this patient's airway?

Effective Communication

– When the patient's clinical status changes, who needs to know and how will this information be disseminated?

– Where is the best place to manage this patient's care?

– What communication tools can assure this patient's safety?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

– Will other specialists be necessary to assure the patient's safety?

A. Preoxygenation

Preoxygenation is essential for decreasing the risk of hypoxia during intubation or sedation. Not only does this fill all alveoli with oxygen before the interruption or alteration of ventilation, it also allows for nitrogen washout and maximizes the time for restoring ventilation, should that become necessary. Oxygen should be provided, ideally utilizing a fraction of inspired oxygen of 1.0 via face mask, for 3 to 5 minutes before administration of any medications. Oxygen flow should then be maintained throughout the procedure. Recent literature suggests that high-flow oxygen via nasal cannula may be used as part of a preoxygenation technique throughout intubation.

B. Premedication

Premedication of some type is required for most intubations and some sedations. As the term implies, these are drugs given before any sedatives in an effort to optimize sedation, avoid pain, and prevent complications. Selection of premedications is based on the individual patient, clinical situation, and sedation goals determined during pre-sedation planning.

1. Anticholinergics

This class of drugs is used for both their vagolytic and secretion-drying properties.

a. Atropine. The most common premedication agent is atropine, a vagolytic that can prevent bradycardia induced by sudden increases in vagal tone. These sudden increases may occur during laryngoscopy, airway manipulation, or with increased airway secretions. Most infants maintain cardiac output via an increase in heart rate rather than an increase in stroke volume. Therefore, any drop in heart rate, especially in unstable patients, may result in a catastrophic drop in cardiac output. According to the 2015 American Heart Association pediatric advanced life support guidelines, atropine (0.02 mg/kg IV/IO; maximum dose, 0.4-1 mg) is a reasonable option in premedication for intubation in patients with preexisting or high risk for bradycardia, especially in those younger than 1 year. This most recent update has done away with recommendations concerning minimum dosing because evidence does not support this practice.

b. Glycopyrrolate. Glycopyrrolate (4 μg/kg IV; maximum dose, 100 μg) decreases secretions to a level similar to that of atropine without consequent central nervous system (CNS) effects. It should be considered in all patients undergoing sedation, especially when using drugs that increase oral or airway secretions (eg, ketamine).

2. Lidocaine

Intravenous lidocaine (1 mg/kg) is known to blunt the transient increases in intracranial pressure (ICP) that occur with laryngoscopy and should be considered for premedication in all patients with presumed or known intracranial pathology. Although its mechanism is unclear, lidocaine administration before laryngoscopy is considered the standard of care in patients with elevated ICP. This agent can also suppress the cough reflex and mitigate bronchospasm, and may therefore be beneficial for intubation of the asthmatic patient. Lidocaine is an antiarrhythmic medication, and its benefits must be weighed against potential side effects in each individual patient. The peak effect of lidocaine is achieved 5 minutes after administration; this needs to be taken into account in planning rapid sequence induction. Additionally, given its modest effects, lidocaine should never delay an emergent intubation.

3. Analgesics

Analgesia is frequently required in premedication, especially before painful procedures in which sedatives without analgesia (eg, propofol) are used. Laryngoscopy should be considered a painful procedure, and the insertion of a laryngoscope is known to cause transient increased ICP. Fentanyl (1 μg/kg) is a good first-line choice because of its rapid onset, short duration, and relatively benign

effect on myocardial activity. Additionally, it blunts sympathetic response to intubation-related stimulation, in turn decreasing spikes in ICP.

C. Sedation

After premedication, the sedative can be given. There are numerous medications, each of which has advantages and disadvantages. All medications given for sedation should have a rapid onset, relatively short duration of action, and minimal side effects. As several of the choices can cause vasodilation and myocardial depression (ie, benzodiazepines, propofol, and barbiturates), adequate intravascular volume must be assured before administration. Similarly, isotonic fluids and vasoactive medications should be immediately available for resuscitation in the event of a rapid drop in blood pressure. Such measures should be at hand when managing patients in shock or with reduced cardiac output. Medications that have no intrinsic analgesia (eg, propofol) must be used in conjunction with adequate analgesia for painful procedures. Each medication has a unique side-effect profile, and selection should only be made after considering the consequences of those side effects in a given patient. Several appropriate choices are usually available in a given clinical scenario, and the agent selected is dependent on the relative balance of positive and negative effects it may, or will, have on a particular patient in a specific situation.

D. Neuromuscular Blockade

Skeletal muscle relaxation, or neuromuscular blockade (NMB), may be required by the clinical situation. Agents may be needed to ensure that the patient remains motionless during a critical procedure, to prevent vocal cord damage during intubation, or to allow ventilation in certain clinical situations. Several studies have shown that use of NMB during intubation can improve airway visualization and lead to a higher likelihood of first attempt success. When needed, these medications have no substitute, but they can also be lethal. If advanced airway management cannot be assured, NMB must be avoided until the airway is secured. In some patients – for example, those with an anterior chest mass – NMB is specifically contraindicated. Each of the paralytic agents has a specific indication and side-effect profile. Careful consideration of the patient, the clinical goal, and the potential side effects of each medication is needed during pre-sedation planning. The dangers associated with these medications cannot be overstated.

V. SPECIFIC SEDATIVE AND ANALGESIC MEDICATION SIDE EFFECTS AND CONTRAINDICATIONS

Every drug used for sedation and analgesia has a specific indication, side-effect profile, and contraindication. Knowledge of these factors is essential before, during, and after a procedure. The risks and benefits of each medication should be weighed during pre-sedation planning, and the agent chosen should be individualized to each patient and clinical situation. Failure to comply can result in avoidable morbidity and death. This overview is not meant to be all inclusive but rather to highlight aspects of several commonly used medications (**Appendix 7**).

A. Nonsteroidal Anti-inflammatory Drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are highly effective analgesics with numerous applications in clinical practice. These medications block inflammation by inhibition of cyclo-oxygenase pathways. In most cases, it is this reduction in inflammation that relieves pain rather than a direct inhibition of pain perception or pain fiber transmission. Because these medications are anti-inflammatory agents, they are also powerful antipyretics. The combined effects make NSAIDs highly desirable in the treatment of postoperative pain. They do not cause respiratory depression, constipation, or itching, making them near-perfect primary or adjunct medications in the treatment of moderate to severe pain. However, several side effects limit their usefulness. First, most NSAIDs can inhibit platelet function either directly or indirectly, leading to an increased risk of perioperative and gastric bleeding. In addition, they can cause, or contribute to, renal failure in patients with preexisting renal insufficiency or other predisposing factors, such as concomitant nephrotoxic medications, liver dysfunction, or heart failure. NSAIDs should be used with caution in these patients. For most patients, the use or addition of NSAIDs to nearly every pain regimen will increase pain control and decrease the dosage of other agents needed to treat that pain.

1. Acetaminophen/paracetamol (10-15 mg/kg every 4-6 hours) is the most commonly used NSAID in pediatric patients. It differs from most NSAIDs in that it is a poor anti-inflammatory agent peripherally, but a powerful anti-inflammatory agent centrally. This makes acetaminophen an ideal antipyretic agent in children because it does not have the associated bleeding and nephrotoxic risks associated with other NSAIDs. In addition, it also appears to have direct central pain-reducing effects independent of its anti-inflammatory properties. Therefore, acetaminophen/paracetamol can be used in conjunction with other NSAIDs to improve pain and fever control via multiple pathways. In hospitalized patients, acetaminophen/paracetamol is available in oral, rectal, and IV formulations.

2. Ibuprofen (10 mg/kg [maximum, 400 mg/dose] every 6 hours) is used frequently to treat fever and pain in children. It is available in numerous tablet sizes, as well as in 20 mg/mL and 40 mg/mL oral liquid preparations, which make this agent particularly useful in children. It is also available as an IV preparation (800 mg/8 mL) approved by the US Food and Drug Administration for infants and children 6 months and older; however, due to the cost, many institutions may not have the drug available.

3. Naproxen sodium (5 mg/kg [maximum, 250-375 mg/dose] every 12 hours) is another oral NSAID effective in treating postoperative pain. It has a longer half-life than ibuprofen and is given every 12 hours instead of every 6 hours. It is a useful alternative to ibuprofen and is available as tablets and an oral suspension (125 mg/5 mL), as well as an IV formulation.

4. Aspirin (salicylic acid) [5-10 mg/kg every 6 hours] was the first NSAID developed. Although an effective agent, its use in most pediatric patients is discouraged secondary to the increased risk of Reye syndrome in those with concurrent viral illness. The “side effect” of platelet inhibition is now the primary reason that aspirin is prescribed. It is the first choice for a daily oral medication for mild anticoagulation in patients at risk for thrombosis, including children.

5. Ketorolac (0.5 mg/kg [maximum, 30 mg/dose] every 6 hours)is a powerful IV NSAID. It has

been shown to be effective in the treatment of postoperative pain even in the absence of adjunctive medications. Concerns of hypersensitivity, renal failure, and increased bleeding in patients younger than 16 years of age have limited its use in pediatrics. However, administration for <5 days in patients with no other contraindications to NSAID use is generally accepted as safe, even in the youngest patients. If concerns exist, consider a single dose immediately before or following a painful procedure.

B. Opioids

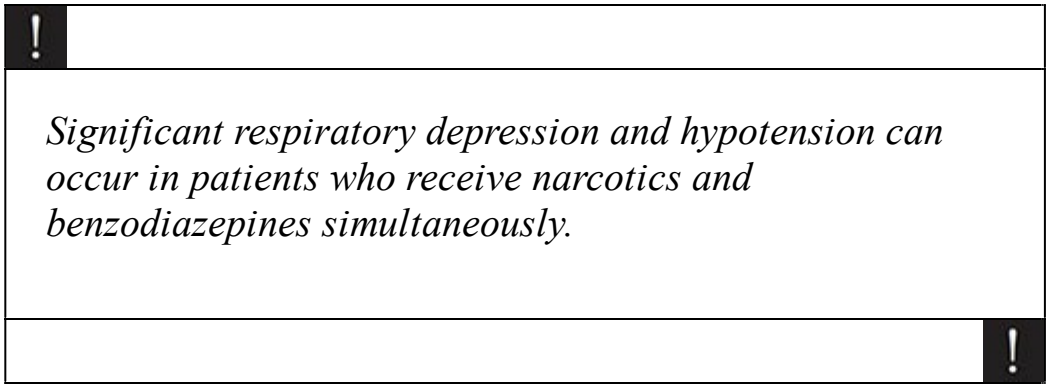
Narcotics are used frequently to prevent pain. All narcotics have similar mechanisms of action. The opioids work as agonists at 3 primary opioid receptors (μ, κ, and δ) with analgesia being mediated at the μ receptor. Each medication blocks transmission of pain fiber signals to the brain and thereby prevents the sensation of pain. In choosing the appropriate narcotic agent to use, the practitioner must first determine the clinical goals:

- Is the agent going to be used for short- or long-term pain management?
- Will a continuous infusion be required, or will intermittent dosing be sufficient?
- Are there specific patient factors to consider?

1. Morphine (0.05-0.1 mg/kg/dose [maximum, 4 mg/dose]) is a good general purpose analgesic. It has a relatively long half-life (2 hours) in most children, but this can increase significantly in small or premature infants. Morphine is metabolized in the liver to generate active metabolites, which are excreted in the urine. For these reasons, morphine must be used cautiously in patients with hepatic or renal dysfunction. Morphine is known to cause vasodilation and hypotension in an immunoglobulin E-independent fashion and should be used with caution in patients with asthma. Additionally, many patients will complain of urticaria due to the associated histamine release. Morphine is also known to cause cardiac depression and should be avoided or used cautiously in patients with low cardiac output or shock. Like all narcotics, morphine can cause significant respiratory depression by decreasing minute ventilation and blunting the CNS response to CO_2. Overall, morphine is a good choice for intermittent or patient-controlled analgesia in those with significant pain. Accumulation of active metabolites and poor clearance in small infants makes it a less desirable agent for continuous infusion in such children.

2. Fentanyl (1-2 μg/kg/dose [maximum, 50-100 μg/dose]) is a synthetic opioid with rapid onset (90 seconds) and clearance ($t_{1/2}$ 30 minutes), but it is stored in fat and can have a significantly longer half-life in obese children. It is 100 times more potent than morphine and must be used at a correspondingly lower dose. Fentanyl is inactivated by the liver and should be used with caution in patients with significant hepatopathy. Like all narcotics, fentanyl causes respiratory depression. Although it does not cause significant myocardial depression, it can cause moderate bradycardia, especially when used with muscle relaxation agents. Unlike morphine, fentanyl does not cause histamine release and is therefore a better choice than morphine for patients with asthma or hypotension. Practitioners should be aware of "rigid chest phenomenon," which can make ventilation difficult or impossible without muscle relaxation. This only occurs with rapid administration at

higher doses, therefore rapid bolus dosing should never be employed. This phenomenon can be mitigated by paralysis with NMB, but advanced airway management will be required. Fentanyl is compatible with most other medications and is therefore a good choice for continuous infusion analgesia. In addition, its fast onset makes it an excellent choice for rapid-sequence intubation or induction.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Significant respiratory depression and hypotension can occur in patients who receive narcotics and benzodiazepines simultaneously.
!

3. Oxycodone and hydrocodone (0.1 mg/kg/dose [maximum, 5-10 mg]) are synthetic opioids. They are used to alleviate moderate to severe pain in patients who can take oral medications. When NSAIDs are inadequate in the relief of post-procedural pain, these oral medications can be considered. As with all opioids, the risk of respiratory compromise should be considered.

4. Codeine (0.5-1 mg/kg/dose [maximum, 30 mg]) is an oral opioid historically used to alleviate moderate to severe pain in patients who can take oral medications. A statement in October 2016 from the American Academy of Pediatrics makes a strong assertion that codeine use in children should be abandoned given its high variability in patient drug metabolism. This metabolic polymorphism has resulted in the respiratory failure of pediatric patients receiving the drug.

5. Methadone (0.1 mg/kg/dose [maximum, 5-10 mg]) is frequently used to treat narcotic withdrawal but can also be used for chronic or intractable pain. It has an extremely long, but frequently variable, half-life (19 hours) and is available in both IV and oral forms. In very small children, the volume per dose in the IV form can be extremely small. Because of its long half-life, the sedative effects of any dosage may not be fully appreciated for 1 or 2 days; therefore, dosage must be adjusted slowly and the effect of that change must be followed for over 24 hours. For these reasons, methadone should be considered in the prevention of narcotic withdrawal, primarily in its oral form, and caution should be used in very small children. There are better choices for “as needed” pain relief and short-term analgesia.

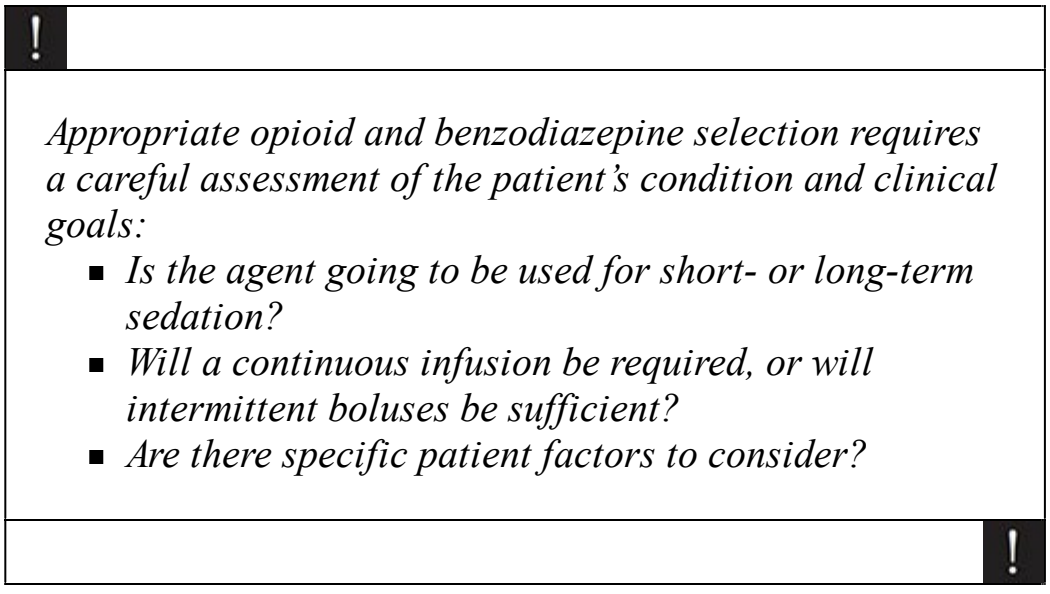
6. Tramadol, (1-2 mg/kg/dose [maximum, 50-100 mg]) a synthetic opioid available in both oral and IV forms (the latter formulation is not available in the United States), binds to opioid receptors and blocks pain transmission using the same mechanism of action that all narcotics do. However, it also blocks the effect of the pain-modifying neurotransmitters serotonin and norepinephrine. Tramadol is rarely used in pediatrics in the United States as it is not recommended by the US Food and Drug Administration in patients younger than 16 years. Tramadol is also known to cause seizures and to lower seizure thresholds in patients who have epilepsy or are taking neuroleptics. For this reason, it must be avoided in these patients. Tramadol is habit-forming and will cause respiratory depression if recommended dosing is exceeded.

7. Meperidine (pethidine) (0.5-1 mg/kg/dose [maximum, 25-50 mg]) is a synthetic opioid with approximately one-tenth the activity of morphine. It has numerous CNS side effects, including seizures and muscle tremors. For these reasons, **it should be always be avoided when other options are available**. There are acceptable alternatives for all indications of this drug.

C. Benzodiazepines

Benzodiazepines are the most commonly used sedatives in critical care. All benzodiazepines increase CNS γ-aminobutyric acid (GABA) receptor activity, causing global CNS depression and consequently sedation. They are excellent first-choice sedatives in clinical situations ranging from anxiolysis to deep sedation. They are also useful anticonvulsants and potent amnestics. However, like opioids, benzodiazepines cause dose-dependent respiratory depression. Unlike opioids, benzodiazepines blunt both hypoxic and hypercarbic respiratory drive. The concurrent use of benzodiazepines and narcotics can result in a profound depression of respiratory drive secondary to the synergistic effect of these medications. This respiratory depression is much more severe than the predicted effect of cumulative doses for the two agents when used independently. In addition, some benzodiazepines can cause substantial myocardial depression and would be contraindicated in patients with borderline cardiac output or shock. As with the narcotics, the appropriate benzodiazepine choice requires a careful assessment of the patient's condition and the clinical goals:

- Is the agent going to be used for short- or long-term sedation?
- Will a continuous infusion be required, or will intermittent boluses be sufficient?
- Are there specific patient factors to consider?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Appropriate opioid and benzodiazepine selection requires a careful assessment of the patient's condition and clinical goals:
Is the agent going to be used for short- or long-term sedation?
Will a continuous infusion be required, or will intermittent boluses be sufficient?
Are there specific patient factors to consider?
!

1. Midazolam (0.1 mg/kg/dose [maximum, 2 mg]) is one of the most common benzodiazepines used. It has a rapid onset and relatively short half-life (2 hours), and is available in oral and parenteral formulations. It may be administered IV, intramuscularly, intranasally and rectally; dosages vary with some routes. It has extensive metabolism in the liver and is eliminated in the urine. Midazolam should be used cautiously in patients with renal or hepatic dysfunction as its half-life can be profoundly increased. Midazolam is a good choice for anxiolysis and long-term sedation by continuous infusion.

In addition, it can be titrated rapidly to treat refractory seizures. Among agents in the benzodiazepine family, midazolam appears to cause the least amount of cardiac depression and is the best choice of a GABA-nergic agent in patients with unclear cardiac output.

2. Lorazepam (0.1 mg/kg/dose [maximum, 2 mg]) has a slower onset than midazolam but has an extremely long half-life (14 hours). For this reason, it is a first-line agent for stopping seizures (**Chapter 15**) and is often used for prolonged sedation. Like midazolam, it undergoes significant liver metabolism and is cleared by the kidneys. With its long half-life, it is a poor choice for use in patients with liver or renal failure. In addition, it is felt to cause more significant cardiac depression than midazolam and should be avoided in patients with persistent shock or low cardiac output. Lorazepam may work well for intermittent dosing in patients requiring long-term sedation. However, it is formulated with propylene glycol and can cause significant lactic acidosis if used for an extended period, especially as a continuous infusion. In addition, its numerous interactions with other medications make it a poor choice for a continuous infusion. Lorazepam is available in both parenteral and oral formulations; oral forms are not typically used in pediatric sedation.

3. Diazepam (0.2 mg/kg/dose [maximum, 10 mg]) has an extremely long half-life. It is extensively metabolized in the liver and should not be used in patients with hepatic or renal failure. It is also formulated with propylene glycol and may cause pain with peripheral IV administration and contribute to lactic acidosis. For these reasons, diazepam is best used as an oral agent, primarily in the amelioration of benzodiazepine withdrawal.

D. Barbiturates

Barbiturates are used much less frequently now than in the past because several agents with less significant side-effect profiles are available. However, barbiturates can be ideal agents in some clinical situations, especially with head injury, elevated ICP, and refractory seizures. All barbiturates are powerful GABA agonists, inducing global CNS depression and concomitant decreased cerebral perfusion. These agents can cause profound respiratory depression, and the need for advanced airway management should be anticipated whenever they are used. All barbiturates cause cardiac depression as well as vasodilation and should never be used in patients with decreased cardiac output, shock, or volume depletion. Individuals with minimal cardiac reserve may become profoundly hypotensive after only a single dose.

1. Pentobarbital (1 mg/kg/dose) is a fourth-line agent used in the treatment of refractory seizures, usually with the clinical intention of temporary coma induction. It has a half-life of nearly 20 hours, and continuous infusion requires advanced cerebral monitoring such as bispectral index or continuous electroencephalography. It can cause neutrophil inactivation and associated increased risk of infection, as well as profound myocardial depression. Pentobarbital should only be used with continuous arterial pressure monitoring. Midazolam infusions have supplanted pentobarbital usage to a large extent, but pentobarbital can still be considered for intubation induction in patients with elevated ICP or for maintenance of medical coma.

2. Phenobarbital (loading dose of 20 mg/kg, which may be given in divided doses; maintenance dosing, 5 mg/kg/day divided every 12 h) is frequently used as a third-line agent in the control of refractory seizures—but it is a second-line agent in patients with refractory seizures who are younger

than 6 months (**Chapter 15**). Phenobarbital has a relatively slow onset but a very long half-life (24-96 hours). Its clinical use should be restricted to the treatment of refractory seizures, treatment of barbiturate dependence, or as an adjunct in difficult-to-sedate patients who are hemodynamically stable.

3. Sodium thiopental (5mg/kg/dose) was formerly the preferred induction sedative in cases of traumatic brain injury with risk for intracranial hypertension. It was selected for its very rapid onset and offset. As with all other barbiturates, it carries the risk of hypotension via both vasodilation and myocardial depression. Similar precautions and patient management measures should be taken. In recent years, thiopental has largely been replaced by propofol. Thiopental is not available in the United States, but is still widely used in the rest of world.

E. Propofol

Propofol (bolus dose, 1-2 mg/kg; continuous infusion, 50-150 µg/kg/min) is an extremely useful short-term medication that avoids many of the problems associated with other drugs. Propofol is unrelated to other general use anesthetics but appears to manifest its activity through the potentiation of CNS GABA receptors, similar to benzodiazepines and barbiturates. Propofol has an extremely rapid onset, is cleared quickly by the liver, and causes very little respiratory depression, making it ideal for short procedures or anesthesia induction. However, like all medications, propofol has several properties that must be carefully considered during pre-sedation planning.

- Propofol induces vasodilation and myocardial depression, which may lead to significant hypotension in dehydrated patients; therefore, it should be avoided in under-resuscitated patients, patients with shock, or those with questionable cardiac output.
- Propofol has been associated with myoclonic movements at low doses but, interestingly, is an effective adjuvant in the treatment of refractory seizures.
- Propofol has no intrinsic analgesic properties and must be used in conjunction with narcotics, such as fentanyl, for painful procedures.
- An increased risk of propofol infusion syndrome is associated with long-term (>48-72 hours) use and high doses (>150 µg/kg/min). This syndrome causes refractory lactic acidosis, cardiac arrhythmia, hypotension, and rhabdomyolysis and can lead to death. The cause is still poorly understood, but some have suggested the disruption of mitochondrial function. If this syndrome is suspected, the propofol infusion should be stopped immediately, even though the syndrome may progress despite cessation.

Propofol is still an excellent drug in several clinical situations. It is ideal for short procedures in relatively healthy individuals (ie, setting/reduction of isolated fractures and central line placement) in conjunction with low-dose narcotics. It should also be considered in the initial treatment of refractory status epilepticus. Similarly, it is an excellent intubation induction agent in patients with traumatic brain injury after adequate resuscitation. Practitioners using propofol should have training and experience in its administration, as well as expertise in airway management, as it may cause profound hypotension, transient apnea, and rapid induction of general anesthesia.

F. Ketamine

Ketamine (1-2 mg/kg/dose IV [maximum, 100 mg], 3-4 mg/kg/dose intramuscular [maximum, 200 mg]) has several properties that make it an ideal agent in certain clinical scenarios. It is an N-methyl-D-aspartate antagonist providing both analgesia and sedation while preserving both blood pressure and respiratory status. It is metabolized by the liver and is secreted with its metabolites in the urine. It has a rapid onset and is quickly eliminated. Ketamine is a sialagogue and may require concurrent use of an anticholinergic agent (ie, glycopyrrolate or atropine) to reduce secretions and prevent laryngospasm. In addition, its profound dissociative properties and resultant emergence phenomenon may require concurrent usage of low-dose benzodiazepines, such as midazolam.

Ketamine is a dissociative anesthetic that has proconvulsant and anticonvulsant properties. It should be used with caution in epilepsy patients. Interestingly, ketamine may be used in cases of refractory status epilepticus. Although it has been reported to cause a transient rise in ICP during infusion, recent studies suggest that it may in fact decrease ICP without lowering blood pressure and therefore maintain cerebral perfusion pressure. These studies suggest that ketamine may be safe to use in patients with both traumatic brain injury and intracranial hypertension. New recommendations around this issue are in process. Ketamine will also cause a transient rise in catecholamine release, which is useful in the maintenance of blood pressure. In addition, this catecholamine surge results in direct β_2-stimulation and consequent bronchodilation. Its airway preserving properties, along with its β_2-stimulation properties, make it an excellent choice for sedation in asthmatic patients. However, its increased α- and β_1-stimulation contraindicate its use in patients with hypertension.

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Ketamine is the drug of choice for intubation of patients with acute severe asthma or septic shock.

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Ketamine is the drug of choice for intubation of children with septic shock; however, its preservation of blood pressure is due to increased release of catecholamines. Therefore, in children with catecholamine depletion, its use may be associated with profound hypotension. Ketamine induces transient horizontal nystagmus, so the staff and parents should be informed of this effect to avoid undue concern or panic. Ketamine should be considered as a first-line agent for intubation of children with asthma and as both short- and long-term sedation in those children. It is also an ideal agent for short, painful procedures in patients with potentially difficult airways but stable respiratory status.

G. Dexmedetomidine

Dexmedetomidine (0.2-1.5 µg/kg/h, IV infusion) is a selective α_2-adrenergic receptor agonist. Its activity is similar to that of clonidine, but with greater potency. It works as a sympatholytic at both central and peripheral receptors. It directly inhibits norepinephrine/noradrenaline release and

produces both analgesia and sedation. It has a rapid onset and is quickly metabolized by the liver to inactive metabolites. Dexmedetomidine has minimal respiratory depression but does decrease heart rate and blood pressure secondary to its sympatholytic activity. Dexmedetomidine must be given by continuous infusion or slow bolus as it can cause transient hypertension due to antagonism of α_1 receptors if given too quickly. It should never be given to patients with atrioventricular node conduction abnormalities, those taking atrioventricular nodal blockers, or patients with bradycardia, hypovolemia, or poor cardiac output as it could worsen those conditions by depleting endogenous catecholamines. With these considerations in mind, dexmedetomidine is increasingly used in pediatrics as a first-line sedative medication that preserves respiratory function well.

H. Etomidate

Etomidate (0.1-0.4 mg/kg/dose, IV) is a sedative hypnotic that can play a role in sedation and rapid sequence induction. It has a rapid onset and offset, making it ideal for use in quick procedures, such as joint dislocation reductions. It also has limited cardiovascular effects, which makes it a good option in rapid-sequence induction of patients with head injury or those with known cardiac disease. Its use has become limited in pediatrics because of some evidence that it may induce or worsen adrenal suppression in young pediatric patients with septic shock. Transient myoclonus is seen in 10% to 15% of patients receiving etomidate, but this has no clinical significance. This side effect should be shared with staff and parents to avoid undue panic.

I. Alternative Administration Routes

A growing body of research demonstrates that many sedatives and analgesics may be delivered transmucosally via the nasal or buccal mucosa. This may provide an alternative to IV administration in situations in which the patient is too anxious or agitated for placement of an IV line. Transmucosal dosing may be adequate for mild sedation or may serve as a way to establish an IV line for more definitive sedation. What follows is a brief guide for more common and effective transmucosally delivered agents. Delivery can be performed via an atomizer-tipped or plain syringe, though atomization allows for better surface area coverage and absorption. For appropriate drug delivery, more concentrated IV solutions should be used for intranasal administration (**Table 20-6**). The maximum recommended volume per naris is 1 mL. The atomizer device has a 0.1-mL fill volume; therefore, doses smaller than 1 mL should have a 0.1-mL overfill added to the syringe to allow for appropriate drug administration.

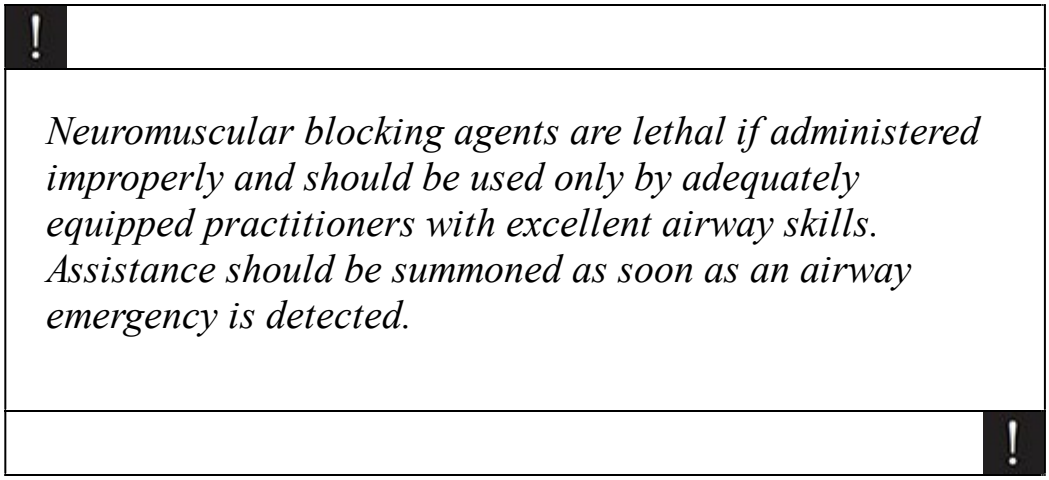
Table 20-6 INTRANASAL ADMINISTRATION OF ANALGESICS AND SEDATIVES

Medication	Dose	Maximum initial dose	Time to onset, min	Duration, min
Fentanyl 50 µg/mL	1-2 µg/kg	100 µg	3-5	30
Midazolam 5 mg/mL	0.2-0.3 mg/kg	10 mg	2-5	30-60
Ketamine 50 mg/mL	6-8 mg/kg	100 mg	3-10	20-60
Dexmedetomidine 100 µg/mL	0.5-2 µg/kg	200 µg	5-10	60

Data taken from Farrington EA. *Pediatric Critical Care in Critical Care Pharmacotherapy*. Kansas City, KA: American College of Clinical Pharmacy; 2016.

VI. SIDE EFFECTS AND CONTRAINDICATIONS OF SPECIFIC NEUROMUSCULAR BLOCKING AGENTS

NMB agents are indispensable in several critical care situations. Skeletal muscle paralysis can prevent vocal cord damage during intubation, maintain absolute immobility during critical procedures, and allow ventilation of patients who cannot be ventilated using deep sedation alone. However, relaxation agents are 100% lethal if advanced airway management cannot be assured. For this reason, if the provider is not completely certain of the ability to maintain a patient's airway after paralysis, relaxation agents should never be given. In addition, NMB does not prevent pain transmission and would cause significant terror and stress in an undersedated patient. For these reasons, paralytics should never be used without adequate concomitant sedation and analgesia. Lastly, non-depolarizing NMB agents, especially in conjunction with steroid usage, can cause a prolonged polyneuropathy of critical illness. This can significantly extend hospitalization and increase morbidity.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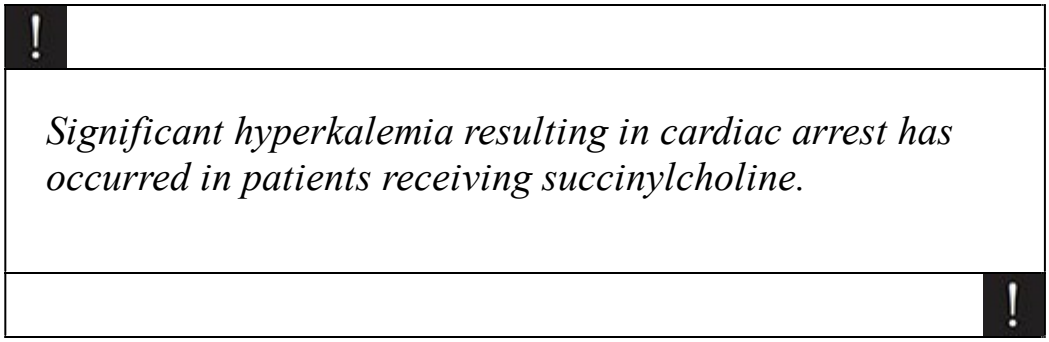
Neuromuscular blocking agents are lethal if administered improperly and should be used only by adequately equipped practitioners with excellent airway skills. Assistance should be summoned as soon as an airway emergency is detected.
!

A. Depolarizing Agents

NMB medications can be classified into two large groups based on their activity at the neuromuscular junction (NMJ). Depolarizing agents open sodium channels at the post-synaptic membrane and cause rapid membrane depolarization, which prevents further neuromuscular transmission. Once the membrane is depolarized, acetylcholine can no longer propagate action potentials throughout the muscle, inducing paralysis.

Succinylcholine (1 mg/kg IV, 3 mg/kg intramuscularly) is the only depolarizing NMB agent in use today. It is an extremely rapid-acting agent (<1 minute) and is completely metabolized by cholinesterases within minutes, independent of liver and kidney function. Its mechanism of action results in a brief period of fasciculation followed by flaccid paralysis. Fasciculation can be blocked by concurrent administration of a partial dose of a non-depolarizing NMB agent. Although its rapid action and short duration are highly desirable, succinylcholine has several side effects, which can be

significant; these include malignant hyperthermia, rhabdomyolysis, bradycardia, hyperkalemia, increased intraocular pressure, and increased ICP. For these reasons, its use is contraindicated in most traumas, neuromuscular disorders, and cases of elevated ICP, glaucoma, significant acidosis, or metabolic derangement. Outside of these conditions, it is an excellent agent, but it should not be used indiscriminately.



B. Non-depolarizing Agents

Unlike the depolarizing agents, the non-depolarizing muscle agents block acetylcholine by binding to its receptor. In this manner, they prevent transmission at the NMJ and induce paralysis until they are metabolized. All non-depolarizing agents have a slower onset than succinylcholine and require a significantly longer period for reversal.

1. Rocuronium (1 mg/kg/dose) has the fastest onset of all non-depolarizing agents at 1 to 2 minutes. It also has the shortest duration of action at about 30 minutes. It has no cardiovascular adverse effects. Although rocuronium is primarily metabolized by the liver, its effects can be prolonged in patients with renal disease. Due to its short duration of action, it is a poor choice for long-term paralysis, but the best substitute for succinylcholine when short-term paralysis is needed (ie, during intubation).

2. Vecuronium (0.1 mg/kg/dose) has an onset of action of 1 to 3 minutes and a duration of action of 30 to 40 minutes. It has no cardiovascular adverse effects. Clearance is significantly reduced in patients with liver disease, and renal dysfunction may also compromise its clearance. Vecuronium is a poor choice for rapid induction, but is probably the best non-depolarizing agent for long-term, continuous sedation.

3. Pancuronium (0.1-0.15 mg/kg/dose) has an onset of action of 2 to 5 minutes and a dose-dependent duration of action of 30 to 60 minutes. A single dose may cause paralysis for more than 2 hours. Most of pancuronium (80%) is eliminated by the kidney, so clearance is significantly reduced in patients with renal dysfunction. Pancuronium has one concerning adverse effect: it has vagolytic properties and can cause significant tachycardia, which may not be well tolerated in some patients. In light of these considerations, pancuronium is only a good choice when vecuronium is not available.

4. Atracurium (0.5 mg/kg/dose) and cisatracurium (0.1 mg/kg/dose) have an onset of action of 2 to 3 minutes and a duration of action of 20 to 35 minutes. Metabolism is by Hofmann degradation. The drug can be used safely in patients with renal or hepatic failure. The rate of degradation is dependent on temperature and pH; therefore, effects may be shortened in children who are febrile or alkalotic, or prolonged in those who are hypothermic or acidotic. Atracurium can cause significant histamine release with resultant hypotension and must be avoided in patients with allergies or asthma. This

release does not occur with cisatracurium. These agents should be considered in patients with significant hepatic or renal dysfunction.

VII. REVERSAL AGENTS

Few drugs are available that can reverse the effects of sedative, analgesic, or NMB agents. However, some agents can decrease morbidity or mortality rates in various clinical situations. They should be immediately available during any sedation, but their usage must be tempered by clinical context.

Naloxone (0.1 mg/kg/dose, up to 2 mg/dose IV; 4 mg/dose intranasally) is a specific antagonist for all opiates. Its action is almost immediate as it displaces opiates from their receptors. All opioid effects are reduced simultaneously at high doses, including respiratory depression and analgesia. At lower doses, naloxone may reverse only respiratory depression and spare analgesia. However, since the medication is usually only given in an emergency, there may not be time to titrate inadequate dosing. Naloxone's half-life is much shorter than that of most narcotics, and it may need to be repeated intermittently until the patient can clear all residual narcotics from the bloodstream. In fact, long-acting narcotics usually require naloxone to be given as a continuous infusion over a prolonged period. Naloxone can be used as an emergency room diagnostic in patients with depressed mental status and poor respiratory effort, but its primary use is reversal of narcotic-induced respiratory depression and avoiding intubation.

Flumazenil (flumazepil), a specific antagonist of benzodiazepines, has a relatively long half-life, approximately 1 hour, but hepatic failure can greatly increase this. Flumazenil completely reverses both the sedative effects and respiratory depression associated with benzodiazepine overdose. Unlike naloxone, flumazenil has several side effects that make it an undesirable medication choice in almost every situation. First, and most importantly, it can trigger seizures in patients with known, or even unknown, seizure disorders. If the patient seizes after flumazenil is given, those seizures cannot be stopped by benzodiazepines, and they may become refractory to second- and third-line medications as well. Second, flumazenil can cause difficult-to-control arrhythmias, tachycardia, and hypertension. Lastly, flumazenil can unmask the effects of tricyclic antidepressant overdose associated with polypharmacy overdose. For all of these reasons, flumazenil should never be used diagnostically for benzodiazepine overdose in any situation. In fact, due to its myriad and severe complications, flumazenil should probably be avoided even in known benzodiazepine overdose. The safer alternative would be to support the patient's airway until the benzodiazepines are cleared. The only exception to this would be a patient with respiratory failure and known benzodiazepine overdose who cannot be ventilated or oxygenated by advanced airway maneuvers.

Non-depolarizing NMB agents can also be reversed to some extent, although not directly, as can opioids and benzodiazepines. Since non-depolarizing agents work by antagonizing acetylcholine binding to the NMJ, high doses of anticholinesterases, such as neostigmine (0.025-0.1 mg/kg/dose), can be used to reverse their effect. However, neostigmine will increase acetylcholine levels at muscarinic receptors as well as the NMJ. In the high doses of neostigmine given to reverse blockade, the patient would exhibit symptoms of acetylcholinesterase toxicity (increased salivation, hyperpyrexia, miosis, and bradycardia) if given without an anticholinergic agent. Either atropine or

glycopyrrolate can be given concurrently with neostigmine reversal doses to prevent these side effects. The time required to reverse paralysis by non-depolarizing agents varies, depending on the percent of acetylcholine receptors occupied by the blocking agent. Therefore, the practitioner must determine that the patient is breathing spontaneously, without assistance, before attempting to remove any advanced airway devices.

Sugammadex sodium (2-4 mg/kg IV; 16 mg/kg IV for immediate reversal of high-dose rocuronium) is a recently developed non-depolarizing NMB reversal agent with action against rocuronium, vecuronium, and pancuronium. It acts to encapsulate the non-depolarizing paralytic molecule through high avidity binding, rendering it inactive. The entire complex is then excreted through the urine. Early studies show it to be safe with few side effects. Widespread use is currently limited by availability and high cost, although use in life-threatening situations when a patient cannot be oxygenated or ventilated would certainly justify the expense.

VIII. TACHYPHYLAXIS AND IATROGENIC WITHDRAWAL

Tachyphylaxis describes the phenomenon of decreased drug effectiveness as it is used for long periods. This is usually the result of increased drug metabolism or downregulation of the receptor of interest. Most medications exhibit this effect, and each patient will respond differently. Infusion rates or dosages required to obtain adequate levels of sedation and analgesia should be expected to increase the longer the patient is exposed to a drug. In fact, tachyphylaxis can be so significant that the practitioner must add agents to an already complicated drug regimen to achieve the desired clinical goal. This is especially important in the continuously paralyzed patient, as sedation and analgesia levels may become inadequate while the patient is still paralyzed. This would result in an awake, paralyzed patient with significant pain.

IX. OPIOID AND BENZODIAZEPINE WITHDRAWAL

A. Pharmacologic Strategies

Opioid and benzodiazepine withdrawal syndromes should be anticipated in all patients requiring ≥5 days of therapy, particularly when synthetic opioids have been administered as a continuous infusion. A long-acting agent, such as lorazepam, can be started 1 to 2 days before anticipated extubation to prevent withdrawal to benzodiazepines; the midazolam infusion can be tapered and stopped gradually once the child is close to extubation. Likewise, methadone can be started at a dosage that approximates the opioid equivalent (while also accounting for pharmacokinetic differences between the 2 agents). It may be initially administered at intervals of 6 hours for approximately 1 day and subsequently at longer intervals. Smaller doses are often effective with careful titration and consultation with a pediatric intensivist or pain specialist. Although methadone is given in 3 to 4 divided doses the first day, the dosing interval is gradually increased and the total daily dose is tapered to avoid drug accumulation. Patients on extended infusions of dexmedetomidine should be

started on clonidine (transdermal or enteral) to avoid withdrawal symptoms.

Lorazepam and methadone are usually tapered on an individualized basis over several days or weeks, depending on the duration of previous treatment with benzodiazepines and opioids. The goal is prevention of physiologically significant complications of withdrawal, such as agitation, seizures, tachycardia, hypertension, diarrhea, and vomiting. Recovery from critical illness and its associated psychological derangements is also expedited by appropriately tapering these agents.

B. Nonpharmacologic Strategies to Control Agitation

The pediatric ICU environment may be upsetting to patients and their families. Units that allow privacy, continuous access to a supportive parent, and controlled sound and lighting may decrease anxiety in young patients. Likewise, promotion of a circadian rhythm—by encouraging exposure to sunlight during the day and quiet, dark conditions at night—may minimize the need to escalate drug therapy. Child life specialists can be helpful in distracting and entertaining patients in ways that permit reduced doses or brief daytime holidays from opioid and sedative infusions.

Key Points

Sedation, Analgesia, and Neuromuscular Blockade

- Sedation, analgesia, and neuromuscular blockade are necessary in caring for critically ill patients but, if used improperly, they can cause significant morbidity or mortality.
- Many of the risks associated with sedation, analgesia, and neuromuscular blockade can be eliminated or reduced by careful pre-sedation planning and preparation.
- Drug choices should be based on the clinical goals and the individual patient. No drug or drug combination is ideal in all situations.
- Each drug has its own side effects and contraindications, which must be carefully weighed against the clinical goals the practitioner is trying to achieve.
- An experienced practitioner should be called for support before sedation is initiated if the clinician is uncertain about the ability to maintain an airway, and to ventilate and oxygenate a patient.
- Always “start low and go slow” when choosing the sedation level.
- Clinicians should recognize their limitations and not initiate sedation when uncomfortable with any patient or scenario. Likewise, they should not use an agent or combination of agents unless confident in doing so.
- The use of reversal agents should be weighed against potentially untoward effects except in life-threatening emergencies.

- Tachyphylaxis should be anticipated with all agents, and dosages should be adjusted at any time.

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INVASIVE MEDICAL DEVICES

Objectives

- Determine the distinguishing characteristics of various tubes and catheters.
- Compare various age-specific catheter and tube sizes.
- Recognize indications for the implementation of various devices.
- Review potential complications associated with the use of invasive medical devices.

Case Study

A teenager who was using a mobile telephone to write a text while driving was injured in a motor vehicle collision. Upon arrival in the emergency department, she is comatose, hypotensive, and hemorrhaging. An endotracheal tube, orogastric tube, subclavian central venous line, chest tube, Foley catheter, and external ventriculostomy are placed to facilitate the patient's resuscitation, stabilization, and care.

Detection

– What is this patient's physiologic status?

– What are the most likely and the most severe injuries?

Intervention

– Which of the presenting injuries require(s) immediate intervention?

– What are the priorities of further assessment and management?

– What additional studies would you like to perform during this patient's evaluation?

Reassessment

– Is the current treatment strategy effective?

– Are cultures, antibiotic coverage, and/or further therapeutic intervention needed?

Effective Communication

– If and when the patient's clinical status changes, who needs to know and how will this information be disseminated?

– What clinical changes might be anticipated?

– Where is the best place to manage the care of this patient?

Teamwork

– How are you going to implement the treatment strategy?

– Who is to do what and when?

After a prolonged ICU stay for traumatic brain injury and multisystem organ dysfunction, during which the patient has undergone ventriculoperitoneal shunt, tracheostomy, gastrotomy, and peripherally inserted central catheter (PICC) placement, she is transferred to a rehabilitation center and remains dependent on technology.

Detection

– What are the most common device complications for which this patient is at risk?

Intervention

– Which of these complications might require immediate intervention?

– What are the priorities of further assessment and management?

– What additional studies would you like to perform during this patient's evaluation admission to the rehabilitation center?

Reassessment

– How often do these devices need to be assessed?

– What would be the first signs of complication for each device?

Effective Communication

– If or when the patient's clinical status changes, who needs to know and how will this information be disseminated?

Teamwork

– How will family be integrated into the care?

– Who is to do what and when?

I. INTRODUCTION

Advances in medical science and technology have helped save and extend the lives of many children. Acutely, invasive medical devices are frequently needed for stabilization and management of critically ill children. In the long term, these devices have become integral to the care of many with complex medical and healthcare needs, allowing many technology-dependent children to receive care outside the hospital and remain within the community. Children with chronic health conditions account for a substantial proportion of cases of severe acute illness. Because of their reliance on technology, all children with invasive medical devices are at significant risk for device-related complications, including displacement, malfunction, and infection, resulting in care interruptions and increased morbidity and mortality rates.

II. TUBES

A. Introduction

Several types of catheters or tubes may be used in the care of critically ill children. They include airways, vascular catheters, gastrointestinal (GI) tubes, peritoneal catheters, internal shunts, bladder catheters. and thoracostomy tubes. These devices provide access to real or potential spaces within the body. Each type has a range of sizes, with the appropriate dimension determined by the child's age, weight, and anatomy. Information regarding artificial airways can be found in **Chapter 2**. Neurosurgical shunts are discussed in **Chapter 15**.

B. Gastrointestinal Tubes

GI tubes are usually made of polyurethane or silicone, have single or double lumens, and may be weighted or vented. The name of the tube generally refers to the anatomical site of insertion and intended use (eg, nasogastric). Typically, tubes are used to provide direct access, to remove GI contents (air, blood, secretions, or ingested toxins), or to instill products (medications, nutrition, or lavage fluids). The indications and advantages of utilizing GI tubes include acute decompression; provision of hydration, electrolytes, and nutrition; medication administration; electrolyte removal with exchange resins; and facilitation of temperature control via lavage fluids. The diameter of a GI tube is measured in French (F) units with each unit equal to 0.33 mm. The pediatric sizes range from 5F to 16F or larger (**Table 21-1**).

Table 21-1 NASOGASTRIC TUBE SIZE

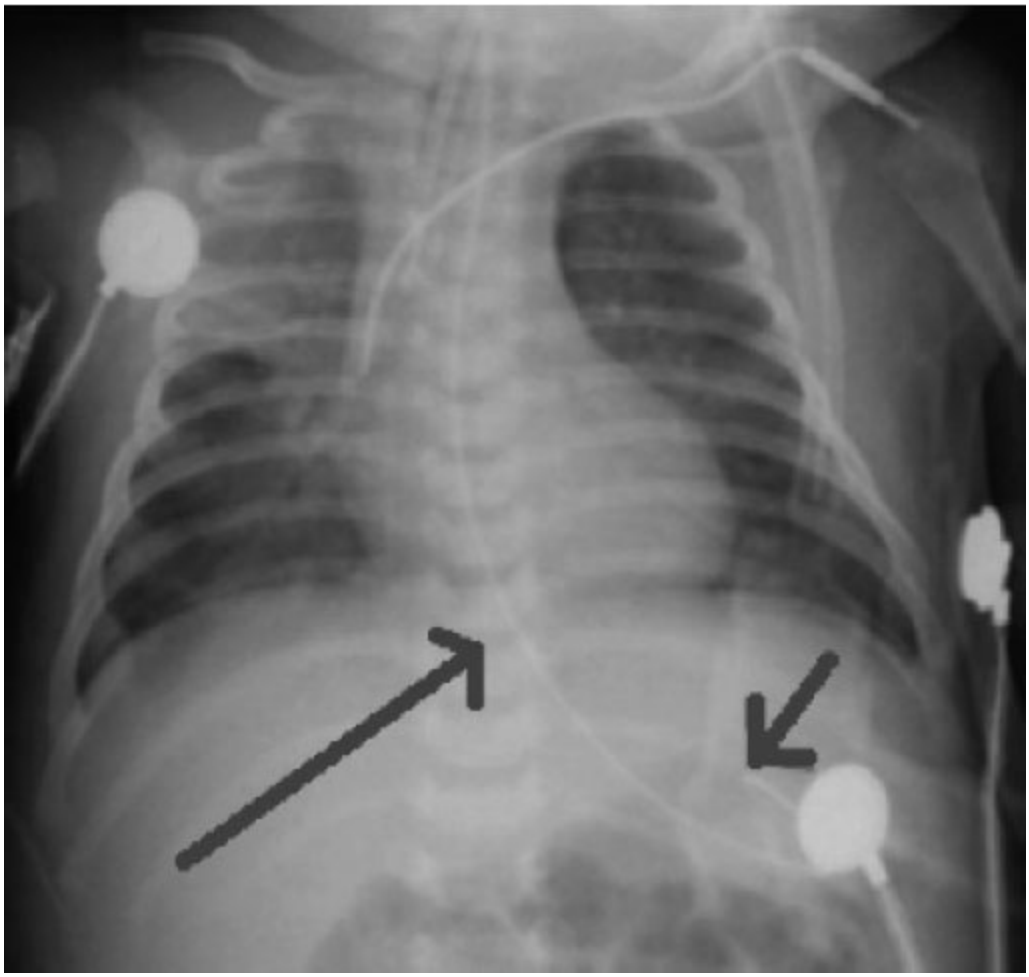
Age	Weight (kg)	Tube Size (F)
0 - 6 mo	3.5 - 7	5 - 10
1 y	10	10
2 y	12	10

3 y	14	10 - 12
5 y	18	12
6 y	21	12
8 y	27	14
12 y	Varies	14 - 16

1. Orogastric and Nasogastric Tubes

Orogastric (OG) and nasogastric (NG) tubes are used for both diagnostic and therapeutic purposes. They are frequently indicated for gastric decompression in patients requiring invasive or noninvasive mechanical ventilator support and for evacuation of gastric contents after an overdose, hemorrhage, or intestinal ileus/obstruction (**Figure 21-1**).

Figure 21-1. Nasogastric Tube



OG placement is performed by passing a tube through the oral cavity, whereas NG tube placement passes the tube inferiorly along the top of the hard palate through the naris, into the nasopharynx. In both instances, the tube is advanced through the pharynx and esophagus into the stomach. OG intubation is specifically recommended for patients with potential head trauma. Potential anterior fossa skull fracture or maxillofacial injury present high risk for inadvertent tube placement into the brain via the cribriform plate or ethmoid bone if inserted nasally.

Nasogastric tube placement is contraindicated in suspected or confirmed anterior fossa skull fracture or maxillofacial injury where the orogastric route is preferred.

The Salem Sump (Covidien AG) is a vented, nonweighted, double-lumen tube commonly utilized in gastric decompression. The second lumen of the tube allows air to irrigate the distal tip of the tube as suction is applied to the other port. Air irrigation decreases the likelihood of gastric irritation from tube adherence to the gastric mucosa. The Salem Sump tube has an anti-reflux valve that prevents the gastric contents from exiting. The traditional Salem Sump is not optimal for feeding because of the stiffness of tubing material and the presence of the venting port; it has more commonly been used for decompression, lavage, or medication administration. However, newer versions of this tube have safe enteral feeding connections.

2. Feeding Tubes

A nonsurgical feeding tube is used to provide enteral or tube feeding when patients are unable to obtain sufficient nutrition by swallowing. Placement is usually temporary for the treatment of acute conditions, but these tubes may be used in chronic conditions. Small-bore, weighted or nonweighted, single-lumen tubes are preferred for enteral feeding. Stylets are sometimes included in the packaging to assist in tube placement, after which the stylet is removed and discarded. For long-term use, small-bore feeding tubes are preferable to larger-bore NG tubes because the risk of complications (sinusitis, otitis, and tissue necrosis at the nares) is lower over time. Small-bore feeding tubes are not designed for drainage of gastric contents. They can be used for administration of nutrients, fluid, and medications either for short-term or long-term use, depending upon the child's needs, prognosis, and family preference. The placement of the distal tip of the tube (NG or transpyloric) will dictate the child's feeding regimen and labelling of the tube. NG placement allows for both bolus and continuous feeding. Transpyloric (nasoduodenal or nasojejunal) feeding tube placement requires nutrition to be provided by continuous infusion only. Bolus feeding should not be infused transpylorically because the duodenum or jejunum cannot accept large-volume delivery of fluid and nutrients (**Figures 21-2** through **21-4**).

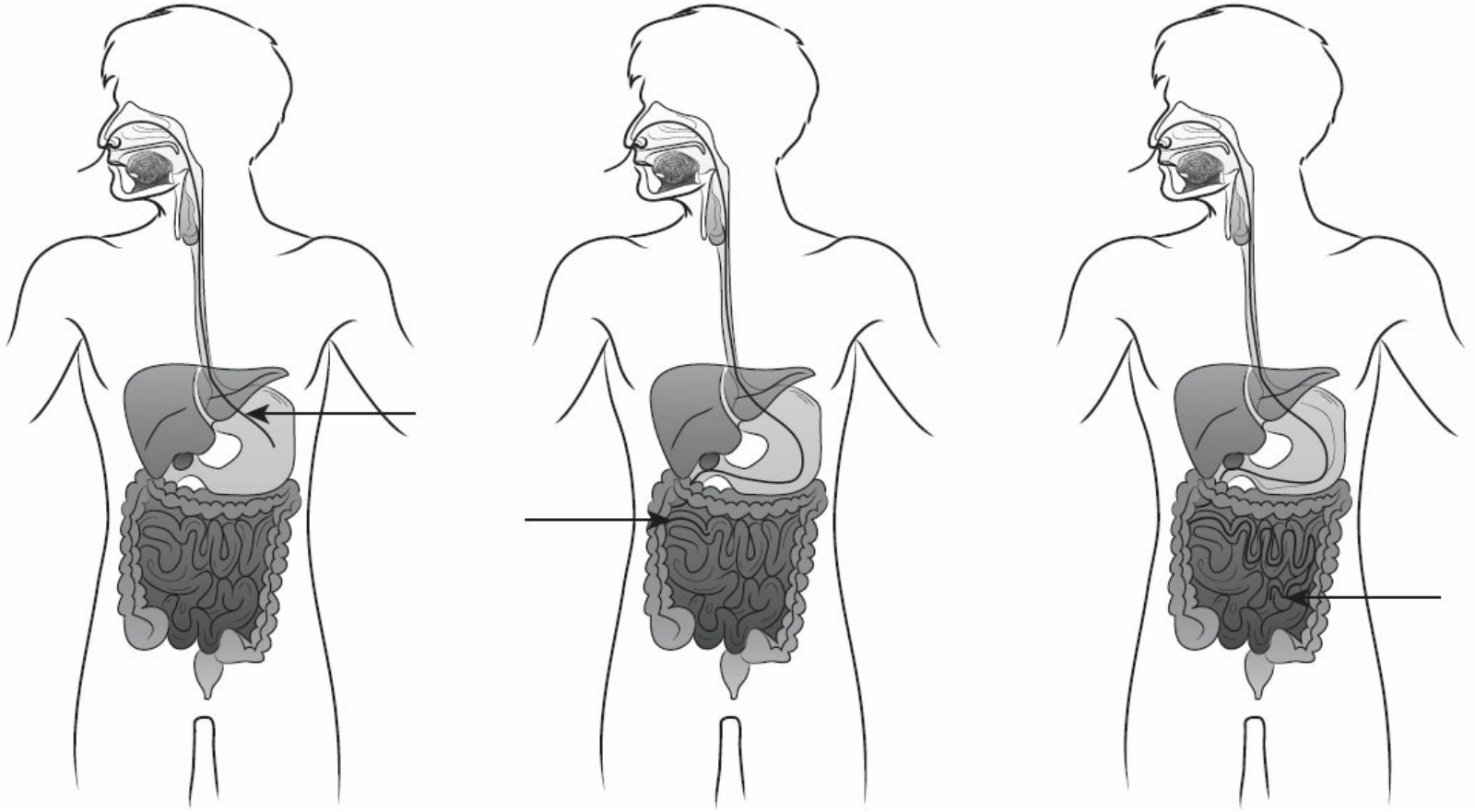


Figure 21-2.*(Left)* Nasogastric Tube Placement

Figure 21-3.*(Center)* Nasoduodenal Tube Placement

Figure 21-4.*(Right)* Nasojejunal Tube Placement

3. Complications

The most frequent complications associated with GI tube placement are listed in **Table 21-2**. If equipment failure is suspected, remove all connecting materials and flush the feeding tube with 5 to 10 mL of water or air. If the tube flushes easily, the equipment likely has not malfunctioned. The inability to flush the tube probably means it is obstructed and needs to be replaced.

Table 21-2	FREQUENT COMPLICATIONS ASSOCIATED WITH SMALL-BORE FEEDING TUBE PLACEMENT

- Coughing, hoarseness, or dyspnea, indicating probable bronchial placement
- Epistaxis
- Tube coiled in esophagus or posterior pharynx
- Esophageal tear or intestinal perforation related to trauma of placement
- Dislodgement or removal by patient
- Aspiration of stomach contents despite appropriate placement
- Clogging of enteral tube with formula or medication fragments
- Skin irritation or breakdown at insertion site
- Pneumothorax from inadvertent pleural placement

C. Surgically Placed Feeding Tubes

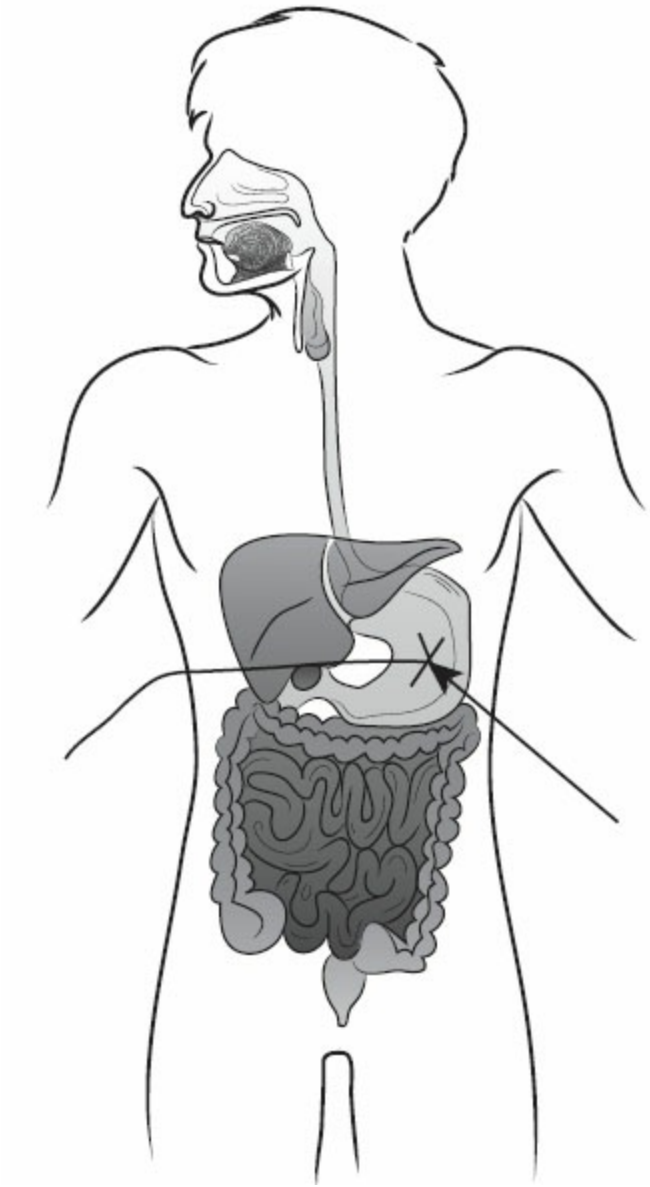
An increasingly large population of children now have surgically placed enteral access tubes. These include gastrostomy, gastrojejunal, and jejunostomy tubes and are used for longstanding needs for artificial nutrition. Such tubes also provide the option to decompress or drain stomach contents. Occasionally, incomplete drainage may occur, and an additional large-bore NG or OG tube may be needed to facilitate full decompression.

1. Gastrostomy Tubes

Gastrostomy or gastric feeding tubes (**Figures 21-5** and **21-6**) are most common and can be placed either surgically (laparoscopic or open laparotomy) or transcutaneously with endoscopic assistance. Use of percutaneous gastrostomy or percutaneous endoscopic gastrostomy (PEG) tubes avoids the need for laparotomy. These tubes initially depend on the traction exerted on them as they are anchored to the skin to maintain contact between the gastric opening and the anterior abdominal wall. PEG tubes often have rigid crossbars within the stomach, which preclude traction removal and require a second endoscopy for removal. These tubes cannot simply be cut and allowed to pass through the GI tract of a young child, as is possible in adults. Small bowel obstruction and esophageal erosion with perforation secondary to esophageal device "reflux" have been reported. Other potential complications include loss of contact between the stomach and abdominal wall, resulting in intraperitoneal soilage and injury to other viscera during placement.

Figure 21-5. Gastrostomy Tube

Figure 21-6. Gastrostomy Tube Placement



Tubes used for laparoscopic placement are either primary low-proifle (MIC-KEY or Passport) tubes or other balloon catheters. These tubes are also anchored to the anterior abdominal wall; however, they are anchored with a balloon. This laparoscopic-assisted procedure avoids laparotomy, while minimizing the risk of injury to adjacent organs (eg, colon) during placement of the gastrostomy tube. The type of catheters used may vary. Operative gastrostomy tubes are generally mushroom tip (Malecot or de Pezzercatheters) which are simply removed by traction (**Figure 21-7**).

Figure 21-7. Mic-Key button

a. Complications

Excessive force should be avoided when replacing gastrostomy tubes, because this may push the stomach away from the anterior abdominal wall and result in a gastric leak into the peritoneal cavity. If any difficulty is encountered in replacing the gastrostomy tube or if initial placement was recent (within 4 to 6 weeks), a contrast study should be obtained to verify correct placement.

If a gastrostomy tube is inadvertently removed, a Foley catheter should be placed to keep the tract open. Simply placing gauze or a dressing over the site may allow it to quickly close spontaneously.

After assessing the thickness of the child's abdomen, a Foley catheter should be selected at a length sufficient to avoid inflating the balloon in the subcutaneous tract (approximately 2-3 cm for most). The catheter should be placed into the stomach wall and the balloon inflated, then gently pulled back snug to adhere to the abdominal wall. The Foley catheter should not be pushed so far that it passes through the pylorus and is inflated in the duodenum; this will result in gastric outlet obstruction and the potential for duodenum rupture. The pylorus is frequently within 5 to 7.5 cm of the site where the tube enters the stomach.

!

A Foley catheter needs to be placed promptly into the gastrocutaneous tract to maintain patency for the patient with a dislodged gastrostomy tube.

!

This also is the basis for evaluating feeding difficulties in a child with a Foley catheter used as a feeding tube. If the catheter is not properly secured to the skin, the stomach will propel the balloon forward and through the pylorus as if it were a bolus of food, resulting in a proximal small bowel obstruction (emesis may be bilious) and gastric, but not diffuse, abdominal distension. These children usually present with excessive leakage around the tube site. The diagnosis is determined by

examining how much tubing is protruding from the site. The problem is resolved by deflating the balloon, pulling the catheter out, replacing it to the proper depth (inserted 2-4 cm), then securing it appropriately with tape. The best solution is a button gastrostomy tube, because this cannot be sucked into the stomach.

2. Gastrojejunal Tubes

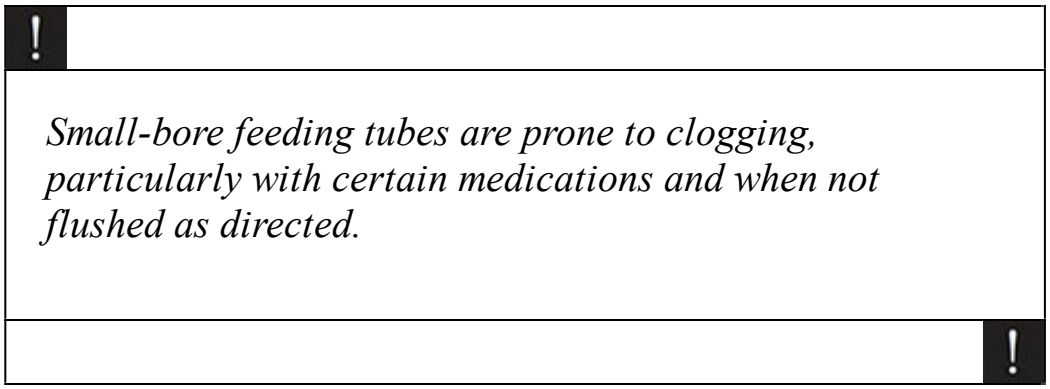
Gastrojejunal tubes are similar to gastrostomy tubes, but close inspection reveals 3 ports: 1 to access the stabilizing balloon, 1 for gastric access, and 1 for jejunal access. Care and placement are much the same as with gastrostomy tubes. During acute use, the gastric port may used for drainage, while the jejunal port is used for feeding and medications. Extra care is needed when administering medications via the gastrojejunal port, as blockage may necessitate endoscopic, fluoroscopic, or ultrasound-guided replacement.

3. Jejunostomy Tubes

A jejunostomy tube is similar to a gastric tube, though it generally has a finer bore and smaller diameter and is surgically inserted into the jejunum rather than the stomach. Jejunostomy tubes are indicated in medical conditions that require bypassing the upper GI tract. These tubes may be used as soon as 12 hours after surgery (**Figure 21-8**).

Figure 21-8. Jejunostomy Tube

Commercially prepared formulas are generally used to provide nutrition and to avoid clogging when used with pump or drip feedings.


!
Small-bore feeding tubes are prone to clogging, particularly with certain medications and when not flushed as directed.
!

D. Thoracostomy Tubes

A thoracostomy tube (also known as a *chest tube, chest drain*, or *pigtail catheter*) is a flexible plastic tube inserted through the chest into the pleural space. These are used to evacuate air (pneumothorax), fluid (pleural effusion, blood, chyle), or pus (empyema) from the intrathoracic space and occasionally for delivery of intrapleural medications (eg, tissue plasminogen activator). Depending upon the child's clinical condition and the urgency of the situation, needle thoracostomy may precede the tube thoracostomy because it may be accomplished quickly.

Chest tubes are sterile, flexible nonthrombogenic catheters made of vinyl or silicone and of varying French sizes and lengths. The size of the tube is dictated by the patient's anatomy and the reason for placement (**Table 21-3**). The insertion site is determined by the indication for the tube. If draining air, the tube is placed anteriorly and toward the apex of the lung; if draining fluid, the tube is directed posteriorly and toward the base of the lung.

Table 21-3 CHEST TUBE SIZE

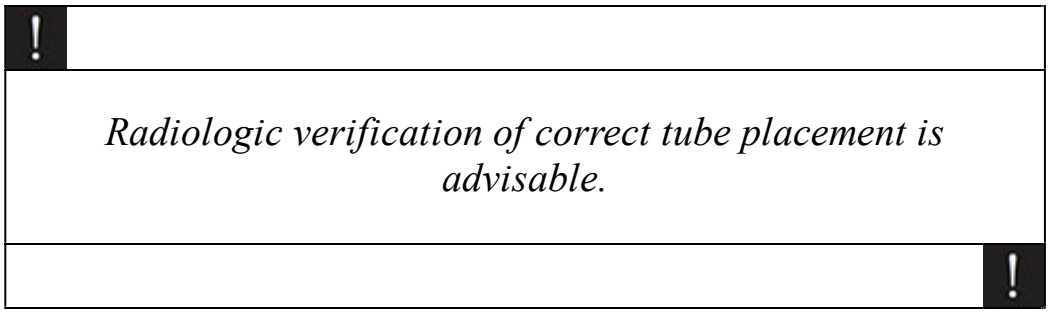
Age	50th Percentile Weight (kg)	Tube size (F)
Newborn	3-5	10-12
Infant	6-9	10-12
Toddler	10-11	16-20
Young child	12-14	20-24
Child	15-22	20-32
Older child	24-30	28-32

1. Complications

The primary complications of tube thoracostomy are similar to those seen with placement of any device through the skin: pain and discomfort, bleeding, and infection. Ironically, two additional

complications of placement secondary to inadvertent lung injury are also indications for chest tube placement: pneumothorax and hemothorax. Less frequent complications include subcutaneous emphysema, thoracic nerve damage, intercostal vascular injury, and organ damage (lung, heart, diaphragm, liver, stomach, and spleen).

Other complications appear to be directly related to tube location after placement. A tube that is not inserted far enough may cause an air leak and subcutaneous emphysema if any of the side drainage holes are outside the pleural space. A tube inserted too far will press against the parietal pleura or thoracic structures, resulting in pain and further tissue injury.



Complications may also occur after the tube is in place. It may become dislodged or disconnected. Pain at the insertion site may cause the child to splint, leading to atelectasis. The skin at the insertion site may become irritated, erythematous, or excoriated, particularly if the occlusive dressing becomes saturated with blood or tissue fluid, or requires frequent changing (**Appendix 11**).

III. CATHETERS

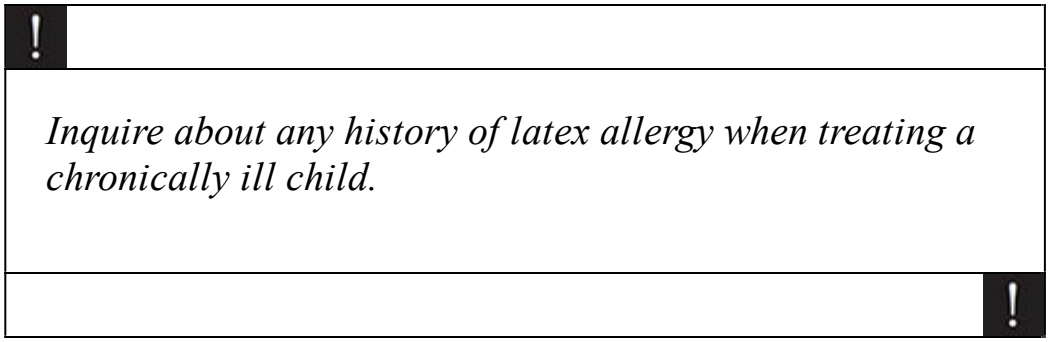
A catheter is a tube that can be inserted into a body cavity, duct, or vessel. These devices allow drainage or injection of fluids and access for surgical instruments. Placement of a catheter (catheterization) may allow for the following:

- Drainage of urine from the urinary bladder
- Drainage of cerebral spinal fluid into a body cavity (**Chapter 15**)
- Administration of IV fluids, medications, or parenteral nutrition
- Direct measurement of pressure in an artery or vein

A. Urinary Catheters

In urinary catheterization, a plastic catheter (eg, Foley catheter) is either inserted into the patient's urinary tract via the urethra and bladder or via suprapubic catheterization. If the catheter is to remain in place for an indefinite time, a balloon at the end is usually inflated with sterile water to keep the catheter from inadvertently slipping out. In this manner, the patient's urine can be collected, measured, and utilized for various medical tests. Catheters come in a large variety of sizes, materials (latex, silicone, polyvinyl chloride, or Teflon), and types (Foley catheter, straight catheter, or coudé tip

catheter). The smallest size is usually recommended for the urethra, although a larger size is sometimes needed to control leakage around the catheter or for continuous bladder irrigation (**Table 21-4**). A large size may also be necessary when the urine is bloody or contains sediment or clots. Individuals with specific medical conditions (spina bifida or urogenital abnormalities), patients who have undergone repeated or prolonged surgeries or mucous membrane exposure to latex, especially early in life, and those with an atopic history or history of food allergy are at a high risk for latex allergies or may have developed allergies or sensitivities to latex after long-term latex catheter use. In such cases, silicone or Teflon types should be used.



Brief catheterization may be used to collect urine specimens for laboratory testing. Patients with neurologic injuries, especially spinal cord injuries, may require long-term clean, intermittent catheterization. A urine collection system is attached to the catheter in circumstances that require the catheter to remain indwelling. The connection of a collecting system maintains a sterile environment.

Table 21-4 URINARY CATHETER SIZES

Age	50th Percentile Weight (kg)	Tube size (F)
Newborn	3-5	5-8
Infant	6-9	5-8
Toddler	10-11	8-10
Young child	12-14	10
Child	15-22	10-12
Older child	24-30	12-14

The primary complication of urinary catheterization is infection. Additional problems include bleeding, tissue trauma, false passage, urethral injury, kidney damage (usually in long-term indwelling use), and development of latex allergy.

B. Indwelling Venous Access Catheters

Indwelling venous access catheters are used to deliver fluid, medication, parenteral nutrition, and blood products directly into a central vein. They may also be used to draw blood samples, and some

are designed specifically for chronic intermittent hemodialysis. A variety of catheters are available for long-term use. The type selected is determined by indications for use and the preference of the patient, family, and healthcare team. The catheters are typically classified by manner of placement and device characteristics.

1. Tunneled Lines

Catheters that exit the skin after being inserted into a large central vein and tunneled beneath the subcutaneous tissue are typically referred to by brand name, such as Broviac (C.R. Bard, Inc.), Hickman (C.R. Bard, Inc.), and Permcath (Covidien AG) [**Figure 21-9**]. These catheters are most often used for long-term parenteral nutrition, chronic medication administration, and chronic intermittent hemodialysis or apheresis. They are secured by means of a Dacron cuff just under the skin at the exit site, which prevents catheter dislodgement and forms a barrier for infection. These catheters may remain in place for extended periods. Insertion is typically done under sedation or general anesthesia, by a radiologist or surgeon. The catheter may have single, double, or triple lumens. External lumens are secured at the exit site with an occlusive dressing. Catheter lumens are typically flushed with an anticoagulant solution on a scheduled basis to prevent blood clots from forming inside the catheter (**Table 21-5**).

Figure 21-9. Broviac Catheter

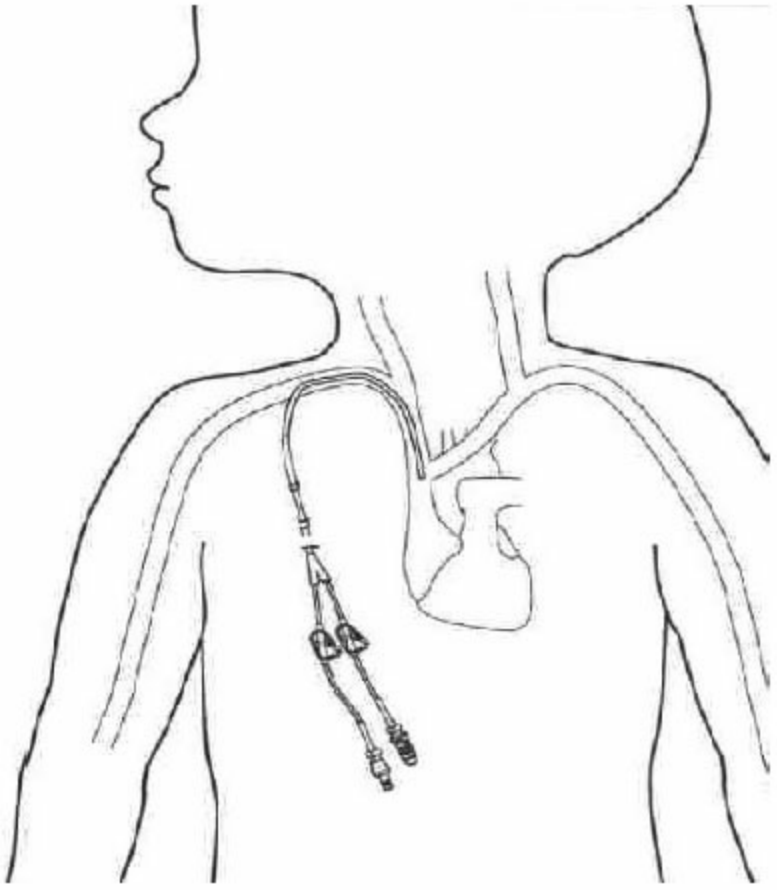


Table 21-5	HEPARIN FLUSH TABLE		
	Description	**Site Care[a]**	**Flush**
Peripheral IV	Range in size	Every 96 h with site rotation and PRN (per	

Catheter	from 24 gauge to 16 gauge. Always use a *T* or *Y* connector.	individual policy)	• Flush amounts for NICU vary. • Flush capped lines every 8 h and PRN. • Use 2-5 mL NS unless heparin flushes ordered.
Central Line Catheter	Any venous catheter in which the tip of catheter is lying in or past the SVC.	Once weekly if transparent dressing; every 48 h if gauze used and PRN. Use central line dressing kit. Do not use chlorhexidine on infants <2 mo. • In NICU, use povidone-iodine x3, then rinse with sterile saline solution, apply dressing. • For other infants <2 mo, use alcohol x3, apply dressing. • All >2 mo, use chlorhexidine x1, apply dressing.	• Flush amounts for NICU vary. • Flush all capped lumens every 12 h and PRN with 2-5 mL 10 U/mL heparin to each lumen. • For blood sampling, discard 2 mL, withdraw specimens, flush with 5-10 mL NS, then heparin per above if capped.
Percutaneous Inserted Central Line Catheter	PICC can be either open-ended or Groshong.	Initial gauze dressing removed in 24 h, then once weekly if transparent dressing; every 48 h if gauze used and PRN. Use central line dressing kit. Do not use chlorhexidine on infants <2 mo; use alcohol x3, then povidone-iodine x3, apply dressing. All >2 mo, use chlorhexidine x1, apply dressing.	• Never use anything smaller than a 10-mL syringe. • Flush amounts for NICU vary. • For all other patients: PICC, 2-5 mL NS, then 2 mL 10 U/mL heparin if <10 kg, 2 mL 100 U/mL if >10 kg, every 12 h to each lumen. • Groshong: 3-5 mL NS every 8 h when not in use, and after each use; 5-10 mL NS after blood infusion or withdrawal. • For blood sampling, only if PICC 3F or larger, discard 2 mL, withdraw specimens, then flush with 5-10 mL NS, then heparin per above if capped.
Tunneled Central Line Cather	Central venous catheters that are tunneled through subcutaneous tissue.	Once weekly if transparent dressing; every 48 h if gauze used and PRN. Use central line dressing kit. Do not use chlorhexidine on infants <2 mo. • In NICU, use povidone-iodine x3, then rinse with sterile saline solution, apply dressing. • For other infants <2 mo, use alcohol x3, apply dressing. • All others >2 mo, use chlorhexidine x1, apply dressing.	• Hickman or Broviac: flush amounts for NICU by physician order. • For all other patients: 2-5 mL NS, followed by 2 mL of 10 U/mL heparin every 24 h. • Groshong: 5 mL NS every 7 days when not in use; 5-10 mL NS after each use. • For blood sampling, discard 2 mL, withdraw specimens, then flush with 5-10 mL NS, then heparin per above if capped.
Implanted Port	Surgically implanted ports have flat septum; percutaneous Groshong port	Change access every 7 d and PRN. Dressing changes once weekly with access changes; every 48 h if gauze used. Use central line dressing kit, new winged infusion set, 20 or 22 gauge, ¾ or 1-inch needle as per patient.	• Never use anything smaller than a 10-mL syringe on ports. • Flush daily with 2-5 mL NS, then 2 mL 10 U/mL heparin if <10 kg, 100 U/mL if >10 kg. At

	has domed septum.	discharge and monthly, use 5 mL of 100 U/mL heparin to all ports. • Groshong port: 5-10 mL NS after each use; 10-20 mL after blood sampling or transfusion. If not in use, flush once monthly with 10 mL NS. May require heparin if valve malfunctions, administer 5 mL heparin 100 U/mL • For blood sampling, discard 3-5 mL, withdraw specimens, then flush with 5-10 mL NS, then heparin per above if capped.

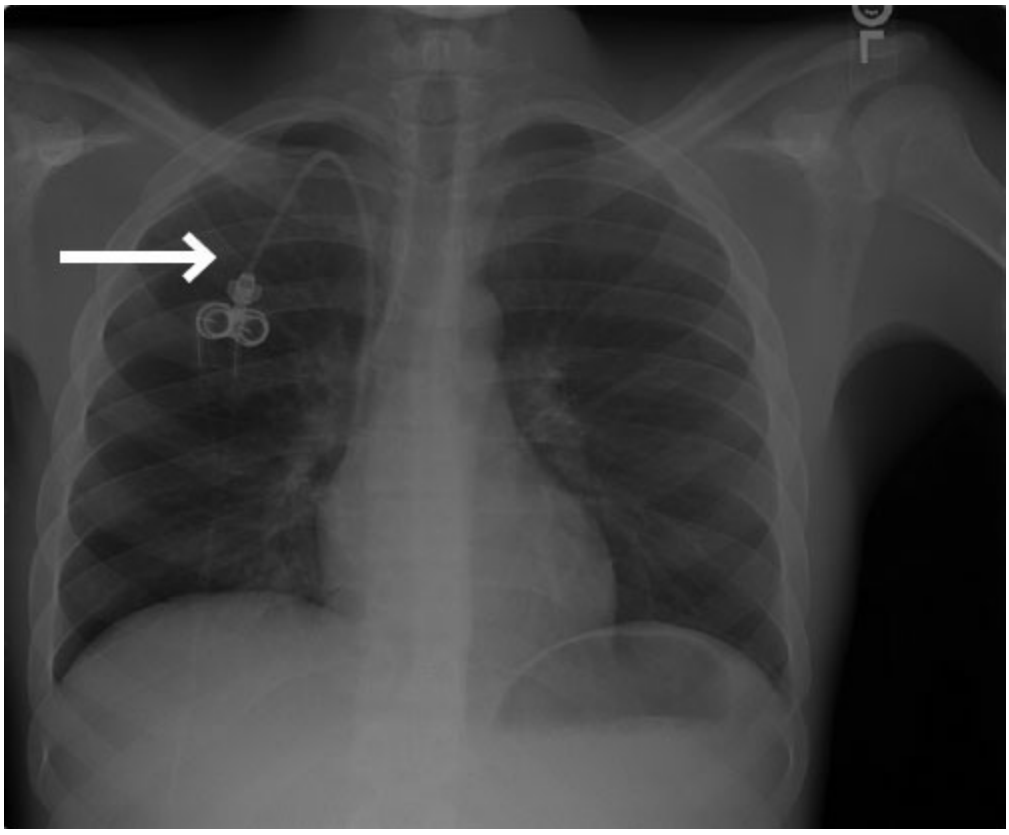
SVC, superior vena cava; PRN, as needed; NICU, neonatal intensive care unit; NS, normal saline; PICC, peripherally inserted central catheter

[a]All IV tubing and caps are changed every 96 h (or as per individual policy). Total parenteral nutrition/lipid tubing is changed every 24 h with solution changes.

2. Ports

Subcutaneous infusion systems require the insertion of a needle through the skin to access a reservoir. The catheter port resides in a subcutaneous pocket in the upper chest, forming a small, palpable bulge under the skin. These catheters, typically Port-a-Cath (Smiths Medical) or PowerPort (C.R. Bard, Inc.), require a flush with anticoagulant only once a month or after completion of access. The risk of infection with this type of catheter is low when not in use. Frequently these ports are used for intermittent central access, such as for chemotherapy administration, intermittent transfusion needs, or difficult vascular access with predictable recurrent need (**Figure 21-10**).

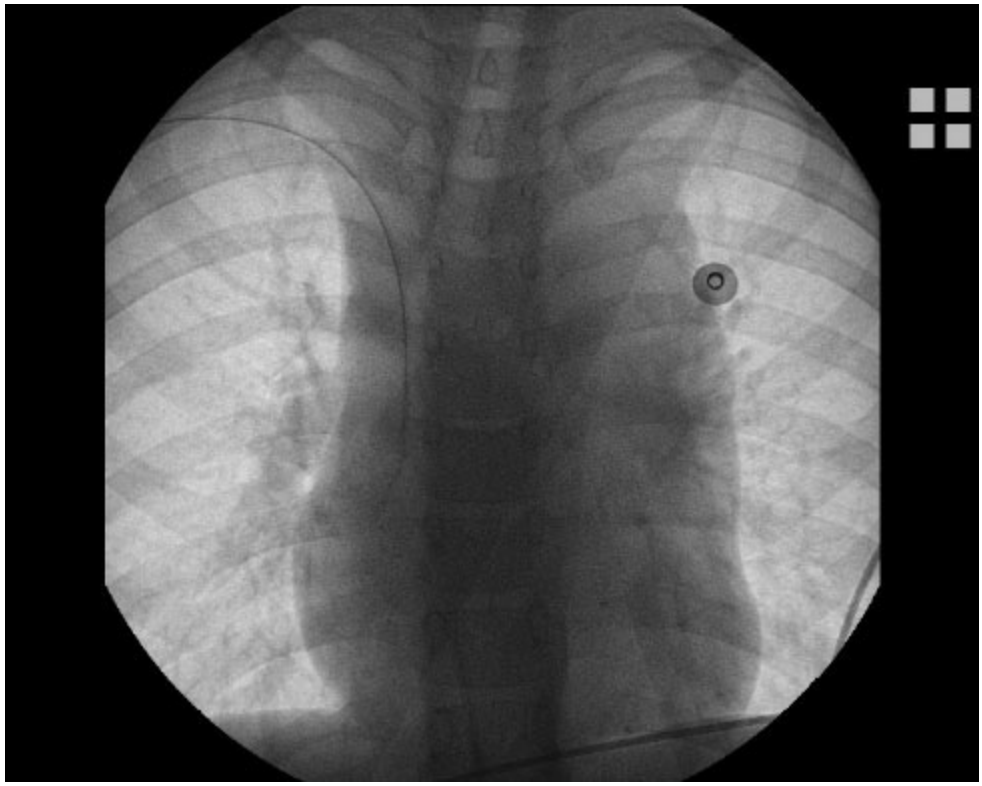
Figure 21-10. Port-a-Cath



3. Peripherally Inserted Central Catheters

A peripherally inserted central catheter (PICC, or PICC line) is a percutaneously placed small flexible catheter typically inserted in the cephalic, basilic, or brachial vein of the arm, traversing centrally (eg, via subclavian) to the superior vena cava until the tip of the catheter rests near the cavoatrial junction. PICCs are usually inserted with the use of local anesthesia or sedation under ultrasound or fluoroscopic guidance and radiographic confirmation of proper placement. A PICC may be used for a prolonged period, typically for chemotherapy, extended antibiotic therapy, or parenteral nutrition (**Figure 21-11**).

Figure 21-11. Peripherally Inserted Central Catheter



4. Complications

Complications associated with indwelling venous access catheters include infection, hemorrhage, occlusion, thrombosis, and phlebitis. These catheters may become displaced, disconnected, dislodged, or obstructed.

Infection is one of the most common complications, especially in a child who is immunocompromised. Fever is a symptom of contamination and infection. The presence of swelling, erythema, tenderness, or bleeding at the exit site also may indicate infection. It is recommended that the child's hemodynamic status be assessed, blood be drawn from the catheter for culture, and a complete blood count be performed.

If the source of the infection is determined, the antibiotic spectrum should be narrowed to best treat the specific organism. Prophylactic antibiotics are not useful. Vigilance with catheter insertion, care, and maintenance has proven to be the best defense against infection. Strict adherence to published insertion and maintenance guidelines is strongly recommended.

Administration of broad-spectrum antibiotics is recommended until the source of infection can be determined.

The catheter may become obstructed by blood clots or by crystals formed by medication or parenteral nutrition. The most serious risk associated with clot formation is the development of pulmonary embolism, which could lead to hypoxemia, respiratory distress, or shock. Signs and symptoms of emboli depend on the organ system affected and may include altered mental status, respiratory distress, cyanosis, dyspnea, chest pain, tachycardia, and shock. Air embolus, another serious risk, has the same signs and symptoms as for a thromboembolism. Treatment involves clamping the catheter, placing the child in a supine position with the left side down and head lower than the body, opening the airway, and administering 100% oxygen. A risk exists for catheter erosion or perforation through the central vein, which may cause pneumothorax, hemothorax, or hydrothorax from infusion of IV fluids; in the event of one of these, prompt decompression may be necessary. Erosion of the catheter tip through the right atrium can result in acute pericardial tamponade.

Bleeding at the exit site may be a presenting sign of an evolving complication. If the catheter is damaged or separated from an external connection, it may be possible to clamp the catheter near the exit site to prevent further bleeding. If the catheter is dislodged, direct pressure should be applied to the site, and ongoing assessment of the child's airway, breathing, circulation, and hemodynamics should be performed to assess for cardiorespiratory compromise.

C. Hemodynamic Monitoring Catheters

Several types of catheters may be inserted to monitor and assess hemodynamic status closely and accurately. These include central venous access catheters and arterial catheters. Because of a reported lack of benefit, the general trend over the past several decades has been to avoid insertion of pulmonary artery catheters or Swan-Ganz catheters in pediatric patients.

1. Central Venous Catheters

Central venous cannulation can be defined as the percutaneous insertion of a vascular catheter within the lumen of a major high-flow vein. The most common insertion sites for access include the internal jugular, subclavian, axillary, and femoral veins (**Table 21-6** and **Appendix 12**). Central venous cannulation provides a more stable and reliable route of venous access than peripheral venous cannulation. It also allows hemodynamic monitoring, sampling of central venous blood for laboratory studies, reliable infusion of vasoactive medications, and the rapid administration of large-volume resuscitation. With a much lower risk of extravasation, central venous access eliminates the complications associated with peripheral administration of potentially caustic medications. Indications for central venous catheter insertion include:

- Resuscitation of circulatory failure
- Administration of vasoactive medications
- Administration of hypertonic solutions
- Administration of caustic chemotherapy
- Measurement of central venous pressure
- Measurement of estimated central venous saturations
- Access for pacemaker or Swan-Ganz catheter placement
- Access for hemodialysis
- Long-term vascular access

Table 21-6 CENTRAL VENOUS CATHETER SIZES[a]

Age	Internal Jugular (F)	Subclavian (F)	Femoral (F)
0-6 mo	3	3	3
6 mo-2 y	3	3	3-4
3-6 y	4	4	4
7-12 y	4-5	4-5	4-5
>12 y	5-7	5-7	5-7

[a]Selection of catheter length depends upon use for monitoring purposes or the need to infuse solutions rapidly.
Data from King C, Henretig FM. *Textbook of Pediatric Emergency Procedures*. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2008.

a. Complications

Complications of central venous catheterization occur more frequently in infants and children than adults (**Table 21-7**). Common complications include pain, infection, thrombosis, vascular perforation, dysrhythmia, air embolus, catheter fragment embolus, and catheter occlusion. Infection can occur with any method or site. In adults, the subclavian location has the lowest incidence of infection. In the pediatric patient, the femoral vein has become the most popular site for central venous cannulation and is associated with a low incidence of infection. The presence of fever, swelling, erythema, tenderness, or bleeding may indicate infection. To prevent infection, maximal barrier precautions must be employed, and stringent cleaning of the catheter insertion site with chlorhexidine should strictly adherence to institutional guidelines for dressing care and maintenance. Prompt evaluation, catheter culture, and narrowing of the antibiotic spectrum to treat the specific organism are essential. Prophylactic antibiotics are not useful. Vigilance with insertion, care, and maintenance has proven to be the best defense against infection.

Table 21-7	COMPLICATIONS OF CENTRAL VENOUS CANNULATION

Site	Complications
Internal Jugular Vein	Carotid artery puncture Carotid artery cannulation Pneumothorax or hemothorax
Right Subclavian Vein	Pneumothorax or hemothorax Tension pneumothorax Thoracic duct puncture Low success rate with inexperience
Left Subclavian Vein	Pneumothorax or hemothorax Tension pneumothorax Thoracic duct puncture Low success rate with inexperience
Femoral Vein	Infection Arterial puncture Failure rate during hypotension/shock Inability to thread central catheter

Data from © 2001 Elsevier. Fleck DA. Central venous catheter insertion (perform). In: Lynn-McHale DJ, Carlson K, eds. *AACN Procedure Manual for Critical Care*. 4th ed. Philadelphia, PA: W.B. Saunders; 2001: 503-513.

Accidental loss of the guidewire into the central circulation is a rare complication that can be avoided by ensuring that the proximal end of the wire is held at all times; if the complication occurs, it may require removal by a interventional radiologist or surgeon. When indications for the device no longer exist, indwelling venous access catheters should be promptly removed. For temporary catheters, the need for continued use should be reconsidered on a daily basis (**Appendix 12**).

2. Arterial Catheters

Arterial cannulation provides a direct means of continuous measurement of blood pressure and pulse, and sampling for evaluation of arterial oxygenation, carbon dioxide tension, and acid-base balance. In addition to these indications, arterial catheter insertion provides accurate pulse pressure monitoring in surgery patients, patients with congenital heart disease, or hemodynamically compromised patients. The most common insertion sites are the radial, femoral, posterior tibial, dorsalis pedis, and axillary arteries (**Table 21-8**). The site selection is based on palpable pulses, overall hemodynamic state, and other anatomic and physiologic factors unique to the individual patient. Because of the distance from the heart and the size of the vessel, the posterior tibial and dorsal pedis arteries are less reliable for hemodynamic pressure monitoring.

Table 21-8	ARTERIAL CATHETER SIZES BY SITE AND PATIENT WEIGHT

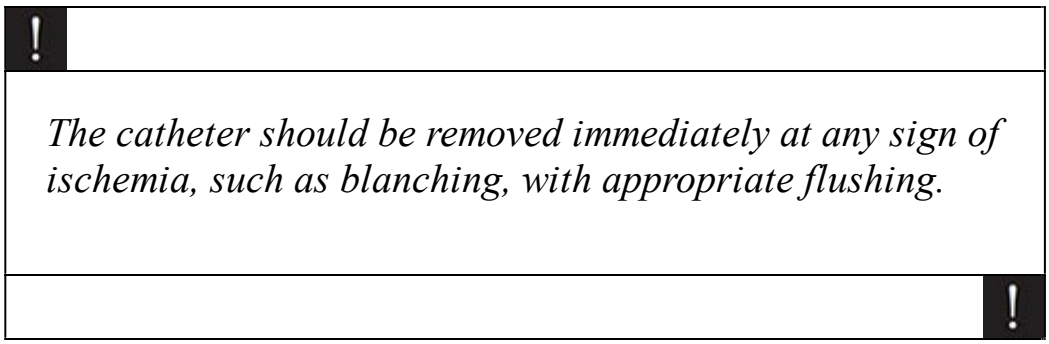
Site	<10 kg		10-40 kg		>40 kg	
	Catheter Size	Size (F)	Catheter Size	Size (F)	Catheter Size	Size (F)
Radial artery, posterior tibial artery, dorsalis pedis	22/24	2.5	22 gauge	2.5/3.0	20/22 gauge	3.0

artery	gauge					
Femoral artery, axillary artery	18/20 gauge	3.0/4.0	16/18 gauge	4.0/5.0	14/16/18 gauge	4.0/5.0

Single-lumen catheters are manufactured in standard French sizes and lengths. Only a single lumen should be used for arterial vessel cannulation.
Reproduced with permission Henretig FM, King C. *Textbook of Pediatric Emergency Procedures*. Baltimore: Williams & Wilkins; 1997. Copyright © 1997 Williams & Wilkins.

a. Complications

Complications of arterial cannulation include arterial insufficiency, arterial thrombosis, air or particulate matter embolization, hematoma, bleeding, pseudoaneurysm, inadvertent nerve injury, and localized or generalized infection Arterial thrombosis may result in tissue necrosis, tissue ischemia, and growth failure in the affected extremity. In children, the most common complications are minor skin lesions, local necrosis, and radial artery occlusion. The potential for problems increases the longer the catheter remains in place. The need for continued use should be routinely assessed daily.



Complications can be minimized by confirming adequate collateral circulation (Allen test) for cannulation of peripheral arteries, selecting an appropriately sized catheter for the artery, performing a careful insertion, exercising vigilant care for the site, and implementing a continuous flush system (**Appendix 15**). The arterial waveform display on the monitor is a safety feature that will help detect catheter occlusion or inadvertent extravasation of blood through an accidentally opened stopcock/3-way tap or break in the system. The extremity with an arterial catheter should be frequently assessed for evidence of ischemia. Placement of an oximeter on the involved extremity/digits can provide a beat-to-beat noninvasive assessment of adequate perfusion via continuous plethysmography. Isotonic (normal saline) flushing, with or without heparin, should be used to maintain arterial line patency. For peripheral arterial catheters, inclusion of papaverine in the flush fluids may prolong catheter life by combating vasospasm.

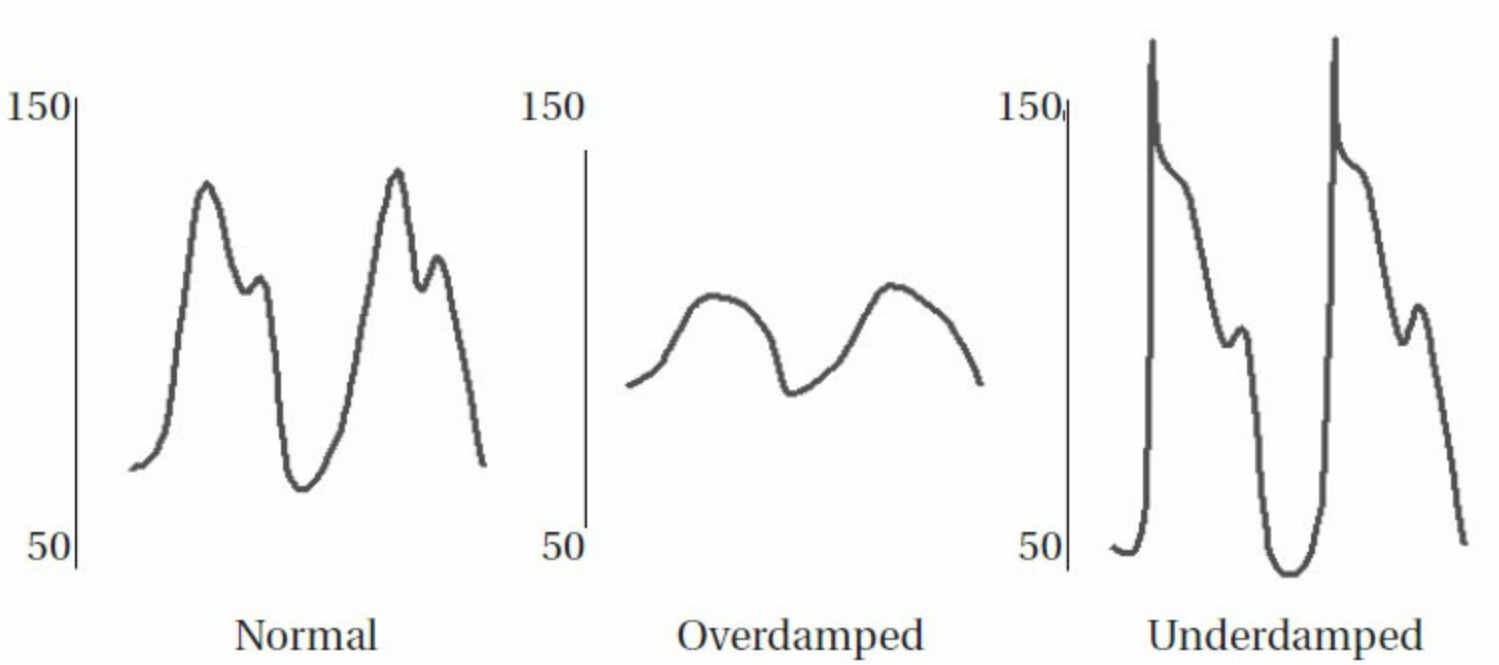
b. Measurements

The arterial pressure obtained from a properly assembled, positioned, and calibrated monitoring system is the "gold standard" of blood pressure measurement. Noninvasive, indirect measurements or manual determination by auscultation of Korotkoff sounds distal to an occluding cuff may vary somewhat from simultaneous direct arterial measurement and are generally slightly lower.

Waveform analysis and troubleshooting are often required to ensure accuracy of measurement. Several technical and anatomic factors may affect accuracy. Waveforms may display fling or

overshoot artifact or a dampened wave. Distortion of the arterial waveform signal may be caused by factors within the blood vessel, hydraulic coupling of the transducer system, and system tubing compliance. Changes in the elasticity of the vessel wall, reflectance of the pulse waveform from the walls or tubing, small air bubbles in the system fluid column, small thrombi around or in the catheter, and stiffness and length of the monitoring tubing are some factors that may result in an incorrect measurement. The arterial blood pressure should also be compared to the noninvasive cuff pressure on a routine basis (**Figure 21-12**).

Figure 21-12. Arterial Wave Forms



Key Points

Invasive Medical Devices

- Children should be examined for the presence of invasive medical devices, which must be evaluated to ensure proper functioning.
- Insertion of invasive devices, especially hemodynamic monitoring catheters, must be performed by individuals who have experience and expertise in the insertion techniques.
- Use of any monitoring or invasive device requires users to be thoroughly familiar with its operation and potential complications.
- The continuing need for invasive medical devices should be assessed periodically; this may be daily for each acute medical device until removed.
- The two primary indications for insertion of an arterial catheter are frequent arterial blood sampling and beat-to-beat blood pressure monitoring. Waveform analysis and troubleshooting are often required to ensure accurate measurements.

- Management of abnormal pleural collections depends upon several factors: the etiology and size of the lesion, the patient's general condition, any associated problems, need for patient transport, and need for mechanical ventilation or general anesthesia. Not all pleural collections require intervention or tube placement.

Suggested Readings

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PEDIATRIC NORMAL VALUES

Table A1-1	VITAL SIGNS: REFERENCE RANGES			
Age Group	**Respiratory Rate (breaths/min)**	**Awake Heart Rate (beats/min)**	**Sleeping Heart Rate (beats/min)**	**Systolic Blood Pressure[a,b] (mm Hg)**
Newborn	30-60	100-180	80-160	60-80
Infant (1-12 months)	30-60	100-160	75-160	72-105
Toddler (1-2 years)	24-40	80-140	80-120	85-102
Preschooler (3-5 years)	22-34	70-120	60-100	89-108
School age (6-12 years)	18-30	65-110	60-90	94-120
Adolescent (13-17 years)	12-16	60-90	50-90	107-132

[a]50th-90th percentile ranges for boys and girls at 50% height indicated

[b]Lower limit of systolic blood pressure by age calculation for children 1-10 years old: 70 + (age in years x 2)

Remember:

- The **patient's** normal range should always be taken into consideration.
- Heart rate, blood pressure, and respiratory rate are expected to increase during times of fever or stress.
- Respiratory rates for infants and children should be counted for a full 60 seconds.
- In clinical decompensation, the child's blood pressure will be the **last** value to change.
- Bradycardia in children is an ominous sign, usually a result of hypoxia. Act quickly.
- Hypotension can be defined as a systolic blood pressure or mean arterial blood pressure below the 5th percentile. In children aged 1 to 10 years at the 50th percentile for height, the predicted 5th percentile systolic blood pressure and mean arterial blood pressure can be quickly determined using the following formulas:

Systolic blood pressure (mm Hg) 70 + (age in years × 2)

Mean arterial pressure (mm Hg) <40 + (age in years × 1.5)

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Table A1-2 LABORATORY TESTS: REFERENCE RANGES

Test	Conventional Unit	SI Unit
Acid Phosphatase[8]		
Newborn	10.4-6.4 U/L	10.4-6.4 U/L
1 month-13 years	0.5-11 U/L	0.5-11U/L
Alanine Aminotransferase (ALT)[1,6]		
Infant	<54 U/L	<54 U/L
Child/adult	1-40 U/L	1-40 U/L
Albumin[1]		
Newborn	3.5-5.4 g/dL	
0-15 days	3.0-3.9 g/dL	
15 day-1 yr	2.2-4.8 g/dL	
1-2 yr	3.6-5.2 g/dL	
3-16 yr	3.9-5.1 g/dL	
3.6-5.2 g/dL		
≥16 yr		
Alkaline Phosphatase[1,6]		
Infant	150-420 U/L	150-420 U/L
2-10 years	100-320 U/L	100-320 U/L
11-18 years, boy	100-390 U/L	100-390 U/L
11-18 years, girl	100-320 U/L	100-320 U/L
Ammonia[1]		
Newborn	90-150 µg/dL	64-107 µmol/L
0-2 weeks	79-129 µg/dL	56-92 µmol/L
>1 month	29-70 µg/dL	21-50 µmol/L
Amylase[5]		
1-30 d	2-16 U/L	
31d-1 yr	0-30 U/L	
1-3 yr	8-79 U/L	
4-9 yr	16-91 U/L	
10-18 yr	19-76 U/L	
Aspartate Aminotransferase (AST)[1,3]		
Newborn	25-75 U/L	
Infant >2 years	15-60 U/L	
Female	13-35 U/L	
Male	15-40 U/L	

Bicarbonate[6]		
Preterm	18-26 mEq/L	18-26 mmol/L
Full term	20-25 mEq/L	20-25 mmol/L
>2 years	22-26 mEq/L	22-26 mmol/L

Table A1-2 LABORATORY TESTS: REFERENCE RANGES (CONTINUED)

Test	Conventional Unit	SI Unit
Bilirubin (Total)[1,6]		
0-1 day		
Preterm	<8 mg/dL	<137 µmol/L
Term	<6 mg/dL	<103 µmol/L
1-2 days		
Preterm	<12 mg/dL	<205 µmol/L
Term	<11.5 mg/dL	<197 µmol/L
3-5 days		
Preterm	<16 mg/dL	<274 µmol/L
Term	<12 mg/dL	<205 µmol/L
>5 days		
Preterm	<2 mg/dL	<34 µmol/L
Term	<1.2 mg/d	L <21 µmol/L
Bilirubin (Conjugated)[1]		
All ages	0-0.4 mg/dL	0-8 µmol/L
Calcitonin[6]		
Newborn	70-348 pg/ml	70-348 pmol/L
Children		
Males	3-19 pg/ml	0.84-5.32 pmol/L
Females	2-14 pg/ml	0.56-3.92 pmol/L
Calcium (Total)[1]		
Preterm <1 week	6-10 mg/dL	1.5-2.5 mmol/L
Full term <1 week	7-12 mg/dL	1.75-3 mmol/L
Child	8-10.5 mg/dL	2-2.6 mmol/L
Calcium (Ionized)[6]		
Newborn <48 hours	4-4.7 mg/dL	1-1.18 mmol/L
Adult	4.52-5.28 mg/dL	1.13-1.32 mmol/L
Carbon Dioxide (CO_2 Content)[6]		
Infant/child	20-24 mEq/L	20-24 mmol/L
Carbon Monoxide (Carboxyhemoglobin)[6]		
Nonsmoker	0-2% of total hemoglobin	
Smoker	4%-9% of total hemoglobin	
Toxic	20%-50% of total hemoglobin	
Lethal	>50% of total hemoglobin	
Chloride (Serum)[6]		

Pediatric	99-111 mEq/L	99-111 mmol/L
C-Reactive Protein[1,6]		
All ages	0-0.5 mg/dL	

Table A1-2 LABORATORY TESTS: REFERENCE RANGES (CONTINUED)

Test	Conventional Unit	SI Unit
Creatine Kinase (Creatine Phosphokinase)[1]		
Newborn	145-1,578 U/L	145-1,578 U/L
Adult female	20-180 U/L	20-180 U/L
Adult male	20-200 U/L	20-200 U/L
Creatinine (Serum)[1]		
Newborn	0.3-1 mg/dL	27-88 µmol/L
Infant	0.2-0.4 mg/dL	18-35 µmol/L
Child	0.3-0.7 mg/dL	27-62 µmol/L
Adolescent	0.5-1 mg/dL	44-88 µmol/L
Fibrinogen[6]		
Newborn	125-300 mg/dL	1.25-3 g/L
All ages	200-400 mg/dL	2-4 g/L
γ-Glutamyltransferase (GGT)[1,6]		
Cord	37-193U/L	37-193U/L
0-3 weeks	0-130 U/L	0-130 U/L
3 weeks-3 months	4-120 U/L	4-120 U/L
>3 months, boy	5-65 U/L	5-65 U/L
>3 months, girl	5-35 U/L	5-35 U/L
1-15 years	0-23 U/L	0-23 U/L
Glucose (Serum)[1,6]		
Preterm	20-60 mg/dL	1.1-3.3 mmol/L
Full term Newborn		
<1 day	40-60 mg/dL	2.2-3.3 mmol/L
>1 day	50-90 mg/dL	2.8-5.0 mmol/L
1 week-16 years	60-105 mg/dL	3.3-5.8 mmol/L
Iron[1]		
Newborn	100-250 µg/dL	18-45 µmol/L
Infant	40-100 µg/dL	7-18 µmol/L
Child	50-120 µg/dL	9-22 µmol/L
Ketones (Serum)[7,8]		
Qualitative	Negative	
Quantitative	0.5-3 mg/dL	5-30 mg/L
Lactate[1]		

Capillary blood		
Newborn	9-32 mg/dL	1.1-3.5 mmol/L
Child	9-22 mg/dL	1.0-2.4 mmol/L
Venous	5-20 mg/dL	0.5-2.2 mmol/L
Arterial	5-14 mg/dL	0.5-1.6 mmol/L

Table A1-2 LABORATORY TESTS: REFERENCE RANGES (CONTINUED)

Test	Conventional Unit	SI Unit
Lactate Dehydrogenase (at 98.6°F, 37°C)[1]		
Neonate	290-1500 U/L	160-1500 U/L
Infant	180-430 U/L	180-430 U/L
Child	110-295 U/L	110-295 U/L
Lead		
Child	<10 µg/dL	<0.48 µmol/L
Lipase[1]		
All ages	4-32 U/L	4-32U/dL
Magnesium[1]		
All ages	1.6-2.4 mEq/L	0.63-1.05 mmol/L
Methemoglobin[6]		
All ages	<0.3 g/dL	<46 µmol/L
Osmolality[1]		
Neonates	as low as 266	
All ages	275-295 mOsm/kg	275-295 mmol/kg
Phosphorus[1]		
Newborn	4.2-9.0 mg/dL	1.36-2.91 mmol/L
0-15 years	3.2-6.3 mg/dL	1.03-2.1 mmol/L
Potassium[6]		
<10 days	4-6 mEq/L	4-6 mmol/L
>10 days	3.5-5 mEq/L	3.5-5 mmol/L
Prealbumin[1]		
Newborn-6 weeks	7-39mg/dL	
6 weeks-16 years	13-42 mg/dL	
Protein (Total)[1,6]		
Newborn-1 month	4.3-7.6 g/dL	
1-3 months	4.6-7.4 g/dL	
4-6 months	4.2-7.4 g/dL	
7-12 months	5.1-7.5 g/dL	
13-24 months	3.7-7.5 g/dL	

25-36 months	5.3-8.1 g/dL
3-5 years	4.9-8.1 g/dL
6-16 years	6.0-7.9 g/dL

Table A1-2 LABORATORY TESTS: REFERENCE RANGES (CONTINUED)

Test	Conventional Unit	SI Unit
Pyruvate[6]		
All ages	0.3-0.9 mg/dL	0.03-0.1 mmol/L
Sodium[6]		
Preterm	130-140 mEq/L	130-140 mmol/L
Older	135-146 mEq/L	135-146 mmol/L
Triglycerides (Fasting)[6]		
Male		
0-5 years	30-86 mg/dL	0.3-0.86 g/L
6-11 years	31-108 mg/dL	0.31-1.08 g/L
12-15 years	36-138 mg/dL	0.36-1.38 g/L
Female		
0-5 years	32-99 mg/dL	0.32-0.99 g/L
6-11 years	35-114 mg/dL	0.35-1.14 g/L
12-15 years 4	1-138 mg/dL	0.41-1.38 g/L
Troponin-I[1]		
0-30 days	<4.8 µg/L	
31-90 days	<0.4 µg/L	
3-6 mo	<0.3 µg/L	
7-12 mo	<0.2 µg/L	
1-18 yr	<0.1 µg/L	
Urea Nitrogen (BUN)[1,2,3]		
Premature	3-25 mg/dL	1.1-8.9 mmol/L
Newborn	2-19 mg/dL	0.7-6.7 mmol/L
Infant/child	5-18 mg/dL	1.8-6.4 mmol/L
Uric Acid[6]		
0-2 years	2.4-6.4 mg/dL	0.14-0.38 mmol/L
2-12 years	2.4-5.9 mg/dL	0.14-0.35 mmol/L
12-14 years	2.4-6.4 mg/dL	0.14-0.38 mmol/L

1. Adapted with permission. © 2015 Elsevier. Engorn B, Flerlage J.. Blood chemistries and body fluids. In: Engorn B, Flerlage J, eds. *The Harriet Lane Handbook*. 20th ed. Philadelphia, PA ;Elsevier; 2015.
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INTRAOSSEOUS NEEDLE INSERTION

I. INDICATIONS

1. Emergency vascular access in children if intravenous (IV) access is delayed for more than 90 seconds or after 1 failed attempt at access.
2. Any fluid, blood product or medication that can be given intravenously can be given by the intraosseous (IO) route in the same dosages.

II. ANATOMY AND PHYSIOLOGY

The bone is supplied by a major artery. A rich venous network in the bone marrow drains into the central circulation. Blood obtained from an IO aspiration can be used to assess serum pH, Pco_2, complete blood count, type and crossmatch, and any electrolyte studies needed. During resuscitation, the onset of action and peak concentrations of medications through the IO route have been shown to be equivalent to the vascular route.

III. EQUIPMENT

1. Needle selection
   - Jamshidi (CareFusion Corp.)
   - Cook Medical Inc.
   - EZ-IO (Vidacare Corp.): http://www.vidacare.com/EZ-IO/Index.aspx
   - FAST1 (Pyng Medical): http://www.pyng.com/products/fast1/
   - Bone Injection Gun (B.I.G.): http://www.actnt.com/BIG/Bone_Injection_Gun.htm

   See Suggested Readings for comparison of these devices.
2. Sterile syringes and infiltrating needles
3. Sterile 4 × 4 gauze sponges
4. Gloves, sterile drapes
5. Skin disinfectant

6. Supplemental oxygen
7. Pulse oximeter
8. Electrocardiography monitor
9. IV tubing, T connector, and fluid

IV. SITE SELECTION FOR INSERTION

1. Proximal tibia at 1 to 2 cm distal to the tibial tuberosity on the flat part of the tibia
2. Distal tibia at 1 cm superior to the medial malleolus on the flat part of the tibia
3. Distal femur at 2 to 3 cm midline above the femoral epicondyles
4. Proximal metaphysis of the humerus
5. Anterior iliac spine
6. Distal radius and distal ulnar

The distal femurs are preferred with infants younger than 2 months old because the proximal tibia is often too thin. If placement is necessary in neonates, insert at proximal tibia, just below the growth plate, distal to the tibial tubercle; in infants 6 to 12 months old, insert 1 cm distal to tibial tuberosity; in children >1 year old, insert 2 cm distal to the tibial tuberosity.

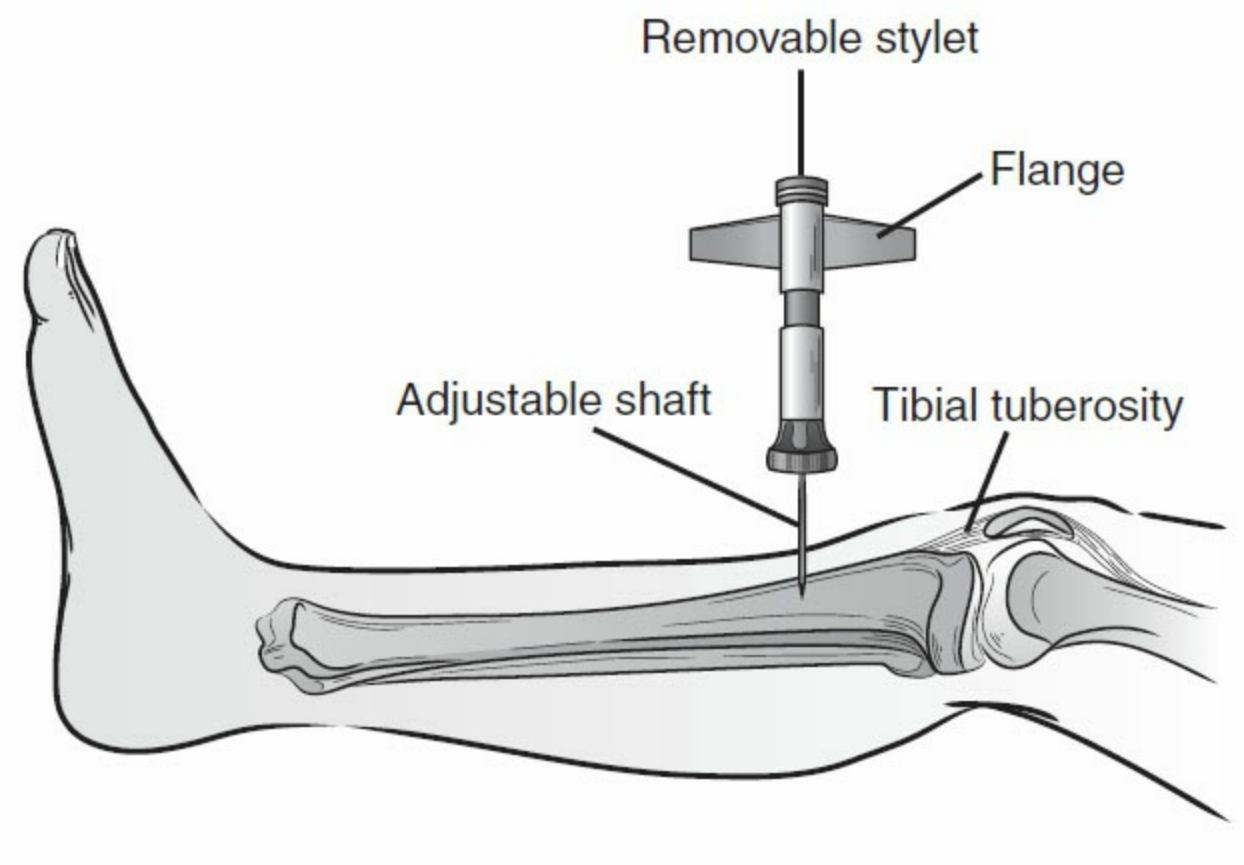
For the older child and adults, in addition to the proximal tibia or distal femur, the area proximal to the medial malleolus, the proximal humerus, or the anterior iliac spine are thin enough for an IO access site. The sternum has been used successfully for adults but is not recommended in children.

V. JAMSHIDI OR COOK TECHNIQUE (TIBIAL SITE)

1. Apply oxygen, monitor pulse oximeter and electrocardiography.
2. Restrain leg with a small sandbag or IV fluid bag behind the knee for support.
3. Disinfect the skin when the time and situation permit. Use povidone-iodine solution or alcohol scrub, 60 seconds of washing and 60 seconds of drying. In children older than 2 months, consider using chlorhexidine, 30 seconds of washing and 30 seconds of drying. To disinfect with a 70 plus percent alcohol solution, 10 seconds of washing and 5 seconds of drying prior to attempting insertion.
4. Infiltrate local anesthetic if warranted by the clinical situation.
5. Use proximal anterior tibia, midpoint of the medial flat surface, 1 to 2 cm below the tibial

tuberosity (**Figure A2-1**).

Figure A2-1. Intraosseous Needle Insertion



6. Insert the needle at 60° to 90° to the skin away from the growth plate; advance with a screwing motion.
7. Use the distal tibia only if the proximal tibia is impenetrable (just proximal [1 cm] to the medial malleolus).
8. Confirm entry into the marrow space by noting a lack of resistance after the needle has passed through the cortex.
9. Aspirate marrow into the syringe; this should be accomplished easily. A properly placed IO needle will allow for marrow to be aspirated in only 50% of placements, so failure to do so does not necessarily indicate improper placement.
10. Infuse isotonic fluids, which should flow freely with proper placement of IO needle.
11. Secure needle by taping flanges to the skin (may require support of external portion of the needle) or by clamping IO needle near skin insertion site with a hemostat and taping the hemostat to the patient's lower leg.
12. Consider flushing with heparin-saline solution.
13. Infuse IV fluids and critical medications (epinephrine, antibiotics, glucose).
14. Observe for infiltration of fluids continuously to prevent injury.
15. Assign a team member to be solely responsible for preventing inadvertent dislodgement of the

IO line until additional secure IV access is obtained.

16. Continue attempts to place IV catheter(s).
17. Discontinue IO infusion and withdraw needle after IV access is established (preferably within 1 to 2 hours) but no longer than 24 hours.
18. Apply pressure to the puncture site for approximately 5 minutes
19. Apply sterile dressing.
20. Do not place the IO needle until medications and fluids are available so as to decrease the risk of dislodgement between placement and infusion.

VI. PRECAUTIONS/COMPLICATIONS

1. Inability to place needle (approximately 20% of patients)
2. Subcutaneous and/or subperiosteal infiltration of fluid
3. Tibia fracture
4. Compartment syndrome
5. Clotting of marrow within the needle
6. Cellulitis, subcutaneous abscess
7. Osteomyelitis (0.6%)
8. Pain (usually minor)

VII. CONTRAINDICATIONS

1. Infection at entry site
2. Burn at entry site
3. Ipsilateral fracture of the extremity
4. Osteogenesis imperfecta
5. Osteopenia
6. Previous attempt at the same site
7. Previous attempt in different location on the same bone

8. Unable to locate landmarks

Suggested Readings

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ACID-BASE BALANCE AND ARTERIAL BLOOD GAS ANALYSIS

Acid-base disorders are common in critically ill children and are due to metabolic and/or respiratory compromise. The arterial blood gas measurement is an important laboratory test in the evaluation of a child's acid-base status.

I. Blood Gas

A blood gas is composed of four basic parts:

1. pH is derived from the logarithmic expression of the hydrogen ion concentration (H^+) and is a measure of acidemia (acidosis) or alkalemia (alkalosis). A normal pH is 7.4 ± 0.05. A pH >7.45 is indicative of alkalemia, and a pH <7.35 indicates acidemia. Respiratory and metabolic processes contribute to the pH as described below; acidemia or alkalemia is the summation of one or more of these processes.

2. $Paco_2$ represents the partial pressure of CO_2 in arterial blood and is a measure of the adequacy of minute ventilation. It has a significant effect on pH in that for every $Paco_2$ change of 10 mm Hg from the baseline of 40 mm Hg, there is a corresponding pH change of 0.08 in the opposite direction. Hence, looking at an arterial blood gas sample, one can determine whether the pH is attributable solely to the $Paco_2$ level.

For example, if a patient has a pH of 7.32 and a $Paco_2$ of 50 mm Hg, this is entirely due to the contribution of the respiratory acidosis (elevated $Paco_2$). However, if the pH is 7.28 and the $Paco_2$ is 50 mm Hg, the $Paco_2$ is only partly responsible for the acidosis, so the patient likely has a mixed/combined respiratory and metabolic acidosis.

3. Pao_2 represents the partial pressure of oxygen in the arterial blood. This provides an assessment of the patient's oxygenation and should be within the range of 80 to 100 mm Hg for a patient who is breathing room air and has no intracardiac right-to-left shunt. In cases of lung disease, hypoxemia might ensue, and Pao_2 levels may decline despite supplemental oxygen.

4. Bicarbonate level (HCO_3^-) is a measure of the presence of metabolic acidosis or alkalosis. Assuming a $Paco_2$ of 40 mm Hg, a change in the bicarbonate level for every 10 mEq/L above or below the normal of 24 mEq/L is associated with a pH change of 0.15 in the same direction. This allows an assessment of the metabolic component contributions to the acid-base (pH) status.

Another corollary that can be drawn from this is the calculation of the base excess (metabolic alkalosis) or base deficit (metabolic acidosis).Because the volume of distribution of bicarbonate is 0.3 L/kg body weight, the total body HCO_3^- deficit can be ascertained: HCO_3^- deficit (in mEq) = (calculated base deficit x 0.3 x weight [kg]).

II. HOW TO EVALUATE AN ARTERIAL BLOOD GAS

When analyzing an arterial blood gas:

A. Determine if the pH is normal (7.35-7.45), low (i.e., acidemia; <7.35), or high (i.e., alkalemia; >7.45).

B. Determine if the $Paco_2$ is high (>45 mm Hg) or low (<35 mm Hg). When **high**, think respiratory acidosis if the corresponding pH is low.

C. Look at the bicarbonate level. If it is low (<22 mEq/L) and the pH is low, think metabolic acidosis. If the bicarbonate level is high (>26 mEq/L) and the pH is high, think metabolic alkalosis.

These examples primarily pertain to acute acidosis and acute alkalosis. If the conditions have been present for several hours or longer, the body attempts to compensate for these abnormalities. Hyperventilation occurs to compensate for the profound metabolic acidosis, as seen in diabetic ketoacidosis; alternately renal compensation ensues in cases of chronic respiratory acidosis, seen in chronic lung disease/bronchopulmonary dysplasia. Many times the abnormalities are mixed, having components of each.

Arterial blood gas analysis is the "gold standard" for measuring acid-base status. In patients with indwelling lines with a free flow of blood, venous blood gas analysis has some benefit, but is different; Pvo_2 levels might not reflect hypoxemia but can provide information on the circulatory status. If the Pvo_2 level is <25 mm Hg, the cardiac output is low. Capillary blood gas analysis is not as helpful due to the potential for hemolysis during sample collection from a heel stick, and the oxygen levels are not predictive of hypoxemia.

Table A3-1 ARTERIAL BLOOD GAS ANALYSIS

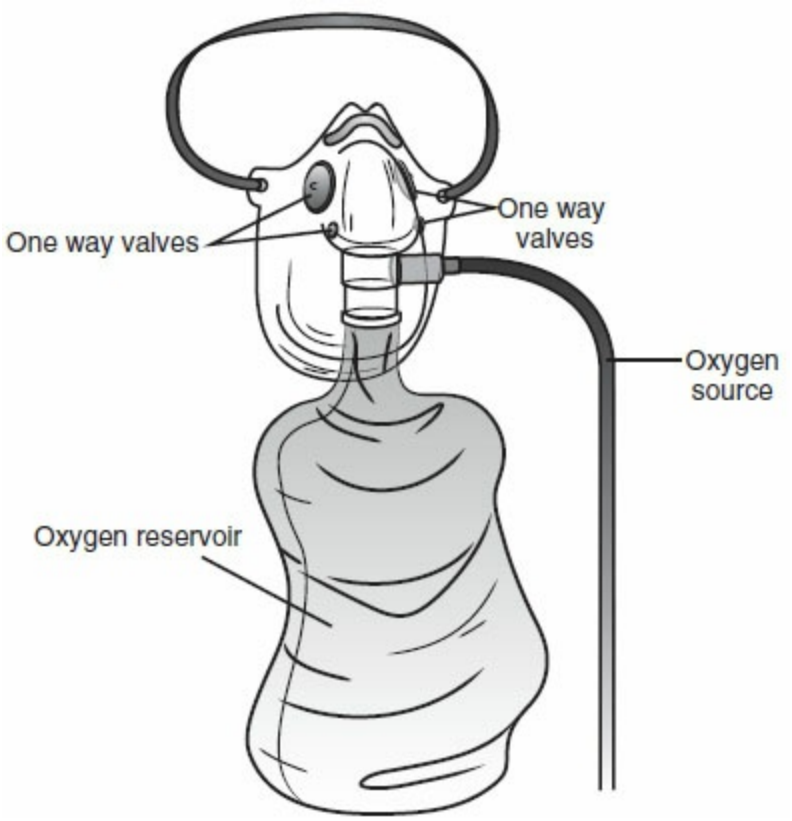
Acidosis	Alkalosis	Metabolic	Respiratory	pH (7.35-7.45)	$Paco_2$ (35-45 mm Hg)	HCO_3^- (22-26 mEq/L)
X			X	<7.35	>45	normal
X		X		<7.35	normal	<22
	X		X	>7.45	<35	normal
	X	X		>7.45	normal	>26

OXYGEN DELIVERY DEVICES

Figure A4-1. Venturi Mask

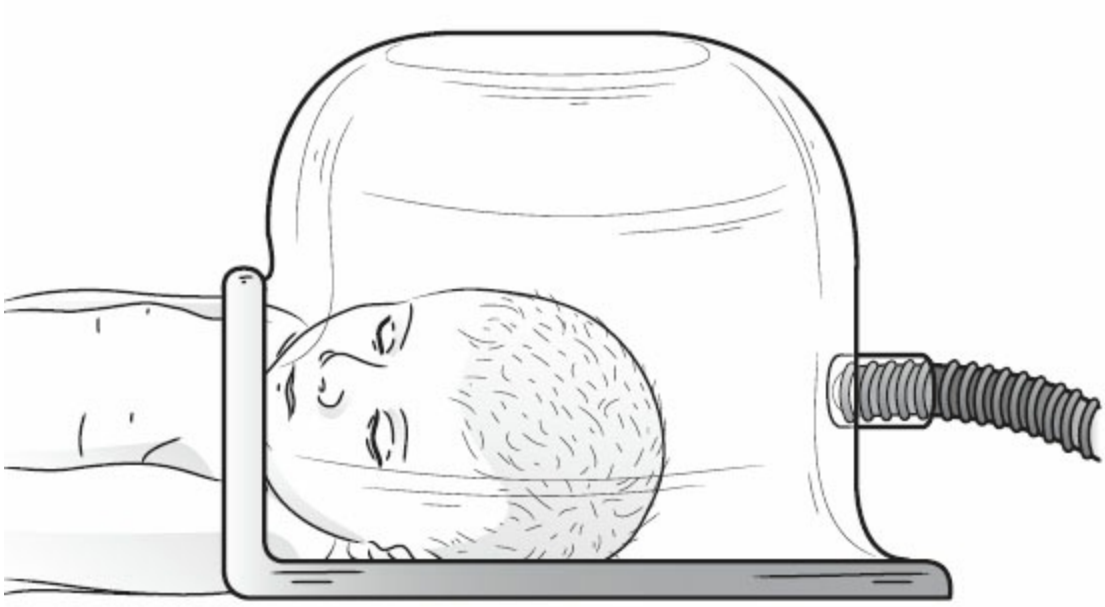
- Oxygen concentration is set by color coded venturi adapters or an air entrainment attached to a humidification cannister.
- Low concentrations = 24%, 26%, 28%, 31%
- High concentrations = 35%, 40%, 50%

Figure A4-2. Oxyhood



- Delivers heated, humidified oxygen to patients weighing <15 kg
- F_{IO_2} is set by dial on blender
- F_{IO_2} range = 21 - 100%
- Minimum flow rate = 10 L/min (to avoid accumulation of exhaled CO_2 from hood)
- In-line oxygen analyzer will provide oxygen concentration.

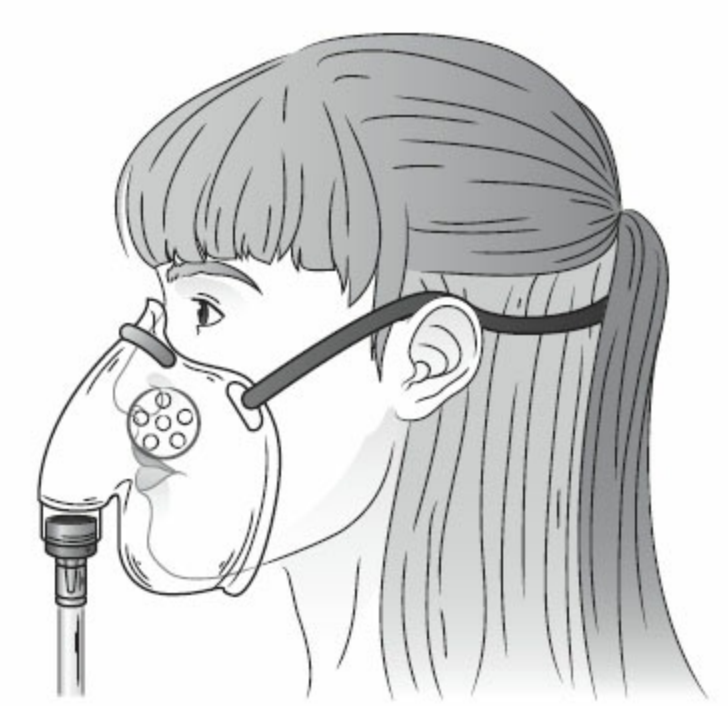
Figure A4-3. Non-rebreathing Mask



- Delivers non-humidified oxygen
- Used to deliver high oxygen concentration
- F_{IO_2} range = 0.6 to 0.9
- Reservoir bag provides 100% F_{IO_2} and minimizes room air dilution

- Flap valves minimize entrainment of room air
- Flow rate is adjusted to keep reservoir bag inflated according to the patient's respiratory pattern

Figure A4-4. Simple Oxygen Mask



- Delivers humidified oxygen
- Minimum flow rate = 6 L/min (to clear exhaled CO_2 from mask)
- Approximate concentrations: 6 L = 0.4; 7 L = 0.5; 8L = 0.6

Figure A4-5. Aerosol Mask

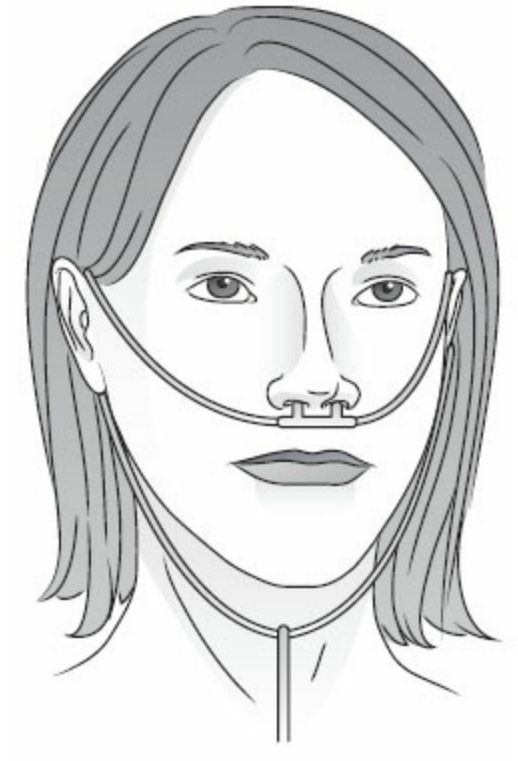
- Delivers aerosolized mist of medication
- Maximum F_{IO_2} = 0.4 to 0.6

- Minimum flow rate = 8 L/min

Figure A4-6. Tracheostomy Mask

- Delivers humidified oxygen or air using oxygen or air (medication nebulizer)
- F_{IO_2} is set by dial on blender
- Maximum F_{IO_2} = 0.4 to 0.6
- Minimum flow rate = 8 L/min

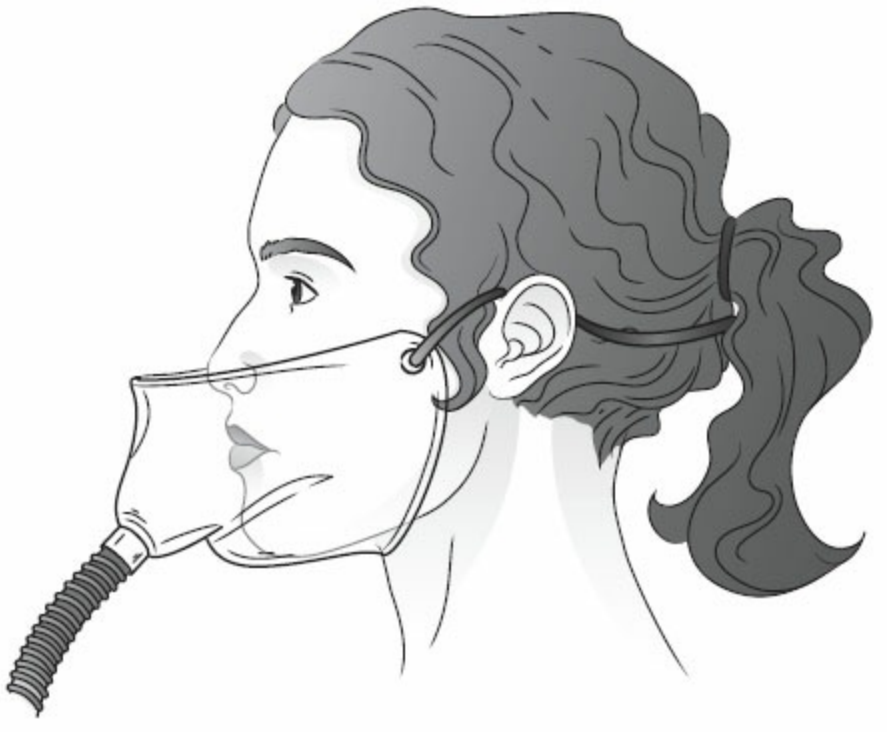
Figure A4-7. Nasal Cannula



- Delivers humidified oxygen
- Nasal passages must be patent
- Oxygen is given by dialing a flow rate
- FIO_2 is variable depending on the respiratory rate and tidal volume of the patient.

Adult	Infant
1 L = 0.24	1/8 L = 0.28
2 L = 0.28	1/4 L = 0.35
3 L = 0.21	1/2 L = 0.45
4 L = 0.36	3/4 L = 0.50
5 L = 0.40	1 L = 0.55
6 L = 0.44	

Figure A4-8. Face Tent



- Delivers cool, humidified oxygen or air
- Loose fit under the chin for patient comfort
- F_{IO_2} is set by dial on oxygen adapter (concentration is unstable)
- Maximum F_{IO_2} = 0.4 to 0.5
- Minimum flow rate = 8 L/min

AIRWAY ADJUNCTS

I. OXYGEN DELIVERY DEVICES

A. *The simple oxygen mask* is a low-flow device that delivers oxygen with a flow rate of 6 to 10 L/min. The oxygen concentration delivered to the patient can reach a maximum of 60% because of entrainment of room air. It is important to maintain an oxygen flow rate of at least 6 L/min to sustain an optimal inspired oxygen concentration and prevent rebreathing of exhaled carbon dioxide.

B. A *partial rebreathing mask* consists of simple face mask with reservoir bag. It provides oxygen concentrations of 50% to 60%. During inspiration, the patient draws gas predominantly from the fresh oxygen inflow and the reservoir bag with minimal entrainment of room air. An oxygen flow rate of 10 to12 L/min is usually required.

C. A *non-rebreathing mask* consists of a face mask and reservoir bag with a valve incorporated into one or both exhalation ports to prevent entrainment of room air during inspiration. Another valve is placed between the reservoir bag and mask to prevent flow of exhaled gas into the reservoir. An inspired concentration of 95% can be achieved with an oxygen flow rate of 10 to 15 L/min and use of a well-sealed mask.

D. A *face tent* is a high-flow soft plastic bucket that is often better tolerated by children than a face mask. Even with a high oxygen flow rate, inspired oxygen concentrations over 40% cannot be reliably provided. A face tent permits access to the face without interrupting oxygen flow.

E. A *nasal cannula* is a low-flow oxygen delivery system that is useful when low levels of supplemental oxygen are required. The net fraction of inspired oxygen depends on the child's respiratory effort, size, and minute ventilation in comparison with the nasal cannula flow.

MANAGEMENT OF DIFFICULT AIRWAY

II. LARYNGEAL MASK AIRWAY

A. Indications

1. Provide an airway and ventilation when bag-mask ventilation is difficult
2. Provide a temporizing airway when endotracheal intubation is unsuccessful

B. Equipment

1. Bag-mask resuscitation unit with high-flow oxygen source
2. Pulse oximeter
3. Electrocardiographic monitor
4. Blood pressure monitoring
5. Gloves, mask, eye protection
6. Laryngeal mask airway (LMA) of appropriate size (**Table A5-1**)
7. Syringe for cuff inflation
8. Water-soluble lubricant
9. Qualitative CO_2 detector or CO_2 monitor
10. Resuscitation cart

Table A5-1 LARYNGEAL MASK AIRWAY SIZE AND CUFF INFLATION

LMA Size	Patient Size	Maximum Cuff Volume	Largest ETT ID (mm)[a]
1	Neonate/infant to 5 kg	Up to 4 mL	3.5
1.5	5-10 kg	Up to 7 mL	4.0
2	10-20 kg	Up to 10 mL	4.5
2.5	20-30 kg	Up to 14 mL	5.0
3	>30 kg/small adult	Up to 20 mL	6.0 cuffed
4	Average adult	Up to 30 mL	6.0 cuffed
5	Large adult	Up to 40 mL	7.0 cuffed

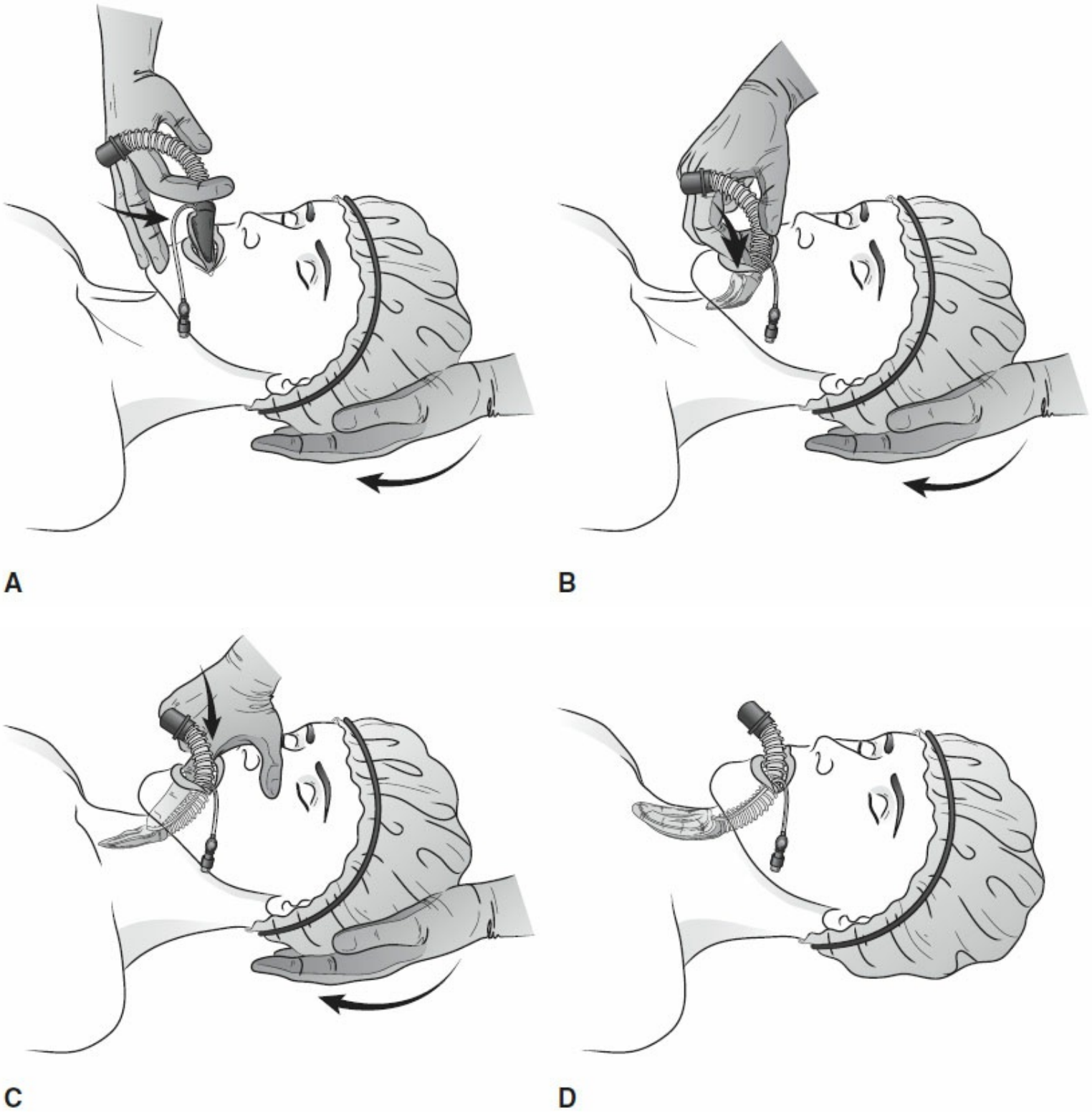
[a]Largest endotracheal tube size (ETT ID) that will fit through laryngeal mask airway (LMA) tube lumen.

C. Preparation for insertion

1. Don gloves, mask, and eye protection.
2. Assure patent airway and optimal oxygenation and ventilation.
3. Assure intravenous (IV) access.
4. Apply pulse oximeter, electrocardiographic and blood pressure monitors.
5. Select appropriate size LMA.
6. Check cuff integrity by inflating and fully deflating.
7. Lubricate only the posterior aspect of the deflated mask with a water-based lubricant.
8. Preoxygenate with 100% oxygen for 2 to 3 minutes, if time permits.

D. Technique (**Figure A5-1**)

Figure A5-1. Insertion Technique for Laryngeal Mask Airway



A) Insert lubricated and deflated mask into the open mouth with the bowl facing anteriorly. **B**) Hold the device like a pencil, pressing against the palate and pharyngeal wall with the index finger. **C**) Continue inserting the cuff behind the tongue into the hypopharynx until definite resistance is felt. **D**) Without holding the device, inflate cuff with enough air to obtain a seal. Attach manual ventilation device and ensure chest movement.

1. The cuff is deflated completely so that it forms a spoon shape and there are no folds in the mask.
2. The operator stands behind the head of the bed, and the bed is raised to a position of comfort for the operator.
3. The patient is placed in the sniffing position (i.e., head extended, neck flexed), unless potential or definite cervical spine injury prevents neck extension.
4. Cricoid pressure is not recommended during placement of the LMA because it may interfere with correct placement.

5. The mask is positioned with the bowl facing anteriorly. Hold the device like a pencil, with the index finger of the dominant hand at the junction of the bowl and tube, pressing against the palate and pharyngeal wall with the index finger.
6. The cuff is inserted into the hypopharynx until definite resistance is felt.
7. Without the operator holding the device, the cuff is inflated with enough air to obtain a seal around the laryngeal inlet. This step results in an outward movement of the tube.
8. The cuff is inflated with enough air to obtain a seal (intracuff pressure of approximately 60 cm H_2O). Maximum volumes are listed in **Table A5-1**, but lesser volume may provide an adequate seal.
9. A manual ventilation device is attached, and chest movement and breath sounds are verified in both lung fields. Correct position should be confirmed with a qualitative or quantitative end-tidal CO_2 detector.
10. If chest movement is inadequate, or if a large air leak is present, the device should be removed and reinserted.
11. When the LMA is positioned appropriately, the tube is secured with tape.

III. ESOPHAGEAL-TRACHEAL DOUBLE-LUMEN AIRWAY DEVICE

Note: The esophageal-tracheal double-lumen airway device is intended for adults and adult-sized children. Pediatric sizes are NOT available.

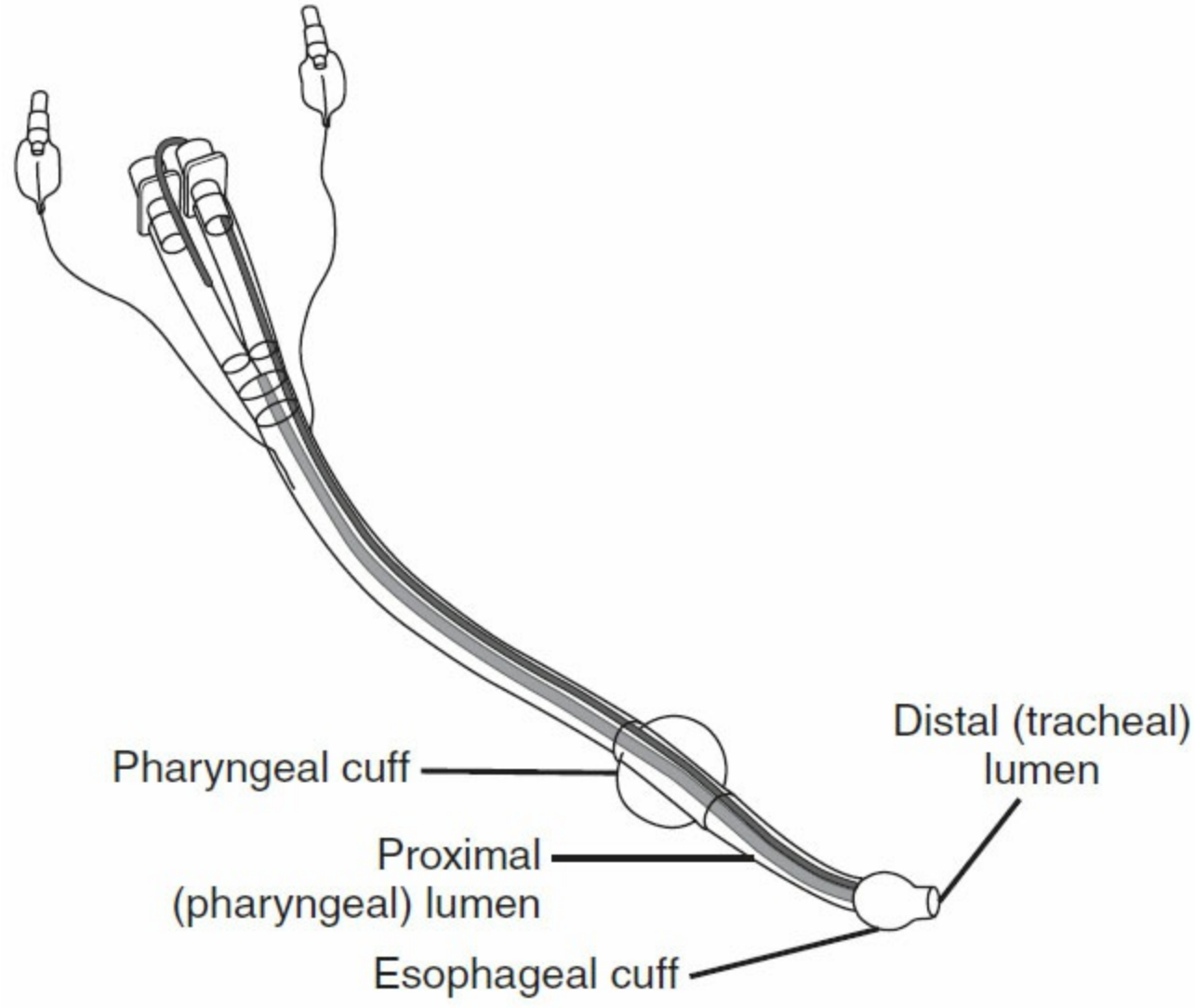
A. Indication

1. Cardiorespiratory arrest and inability to provide an airway by other means

B. Equipment

1. Bag-mask resuscitation unit with high-flow oxygen source
2. Pulse oximeter
3. Electrocardiographic monitor
4. Blood pressure monitoring
5. Gloves, mask, eye protection
6. Esophageal-tracheal double-lumen device (**Figure A5-2**)
7. Syringe for cuff inflation
8. Water-soluble lubricant
9. Qualitative CO_2 detector or CO_2 monitor
10. Resuscitation cart

Figure A5-2. Esophageal-Tracheal Double-Lumen Airway Device



Some tubes have two pilot balloons to allow for independent inflation of the pharyngeal and esophageal cuffs, whereas other tubes have a single pilot port and simultaneously inflate both cuffs. Detection of end-tidal CO_2 in the proximal lumen suggests that the tube is in the esophagus. In the rare instance that the tube enters the trachea, ventilation is only possible via the distal lumen and end-tidal CO_2 will not be detected from the proximal lumen.

C. Preparation for insertion

1. Don gloves, mask, and eye protection.
2. Assure patent airway and optimal oxygenation and ventilation.
3. Assure IV access.
4. Apply pulse oximeter, electrocardiographic and blood pressure monitors.
5. Select appropriate size device. *The esophageal-tracheal double-lumen airway device is intended for adults and adult-sized children. Pediatric sizes are not available.*
6. Check integrity of both cuffs by inflating and fully deflating.
7. Preoxygenate with 100% oxygen for 2 to 3 minutes if time permits.

D. Technique

1. The cuffs should be deflated completely.
2. The operator stands behind the head of the bed, and the bed is raised to a position of comfort for the operator.
3. The patient is placed in a neutral or sniffing position (i.e., head extended, neck flexed), unless

potential or definite cervical spine injury prevents neck extension.

4. The patient's tongue and jaw are grasped between the thumb and index finger, and the device is inserted blindly. It is advanced until the placement ring markers on the tube are positioned as indicated by the manufacturer. Do not force the tube if resistance is met. A laryngoscope can be used to assist with placement.
5. The pharyngeal cuff is inflated first to seal the posterior pharynx.
6. The distal cuff is then inflated.
7. Ventilation should be attempted first through the pharyngeal lumen, and the chest should be auscultated for breath sounds and observed for movement. The tube enters the esophagus approximately 95% of the time.
8. If breath sounds are absent, ventilation should be attempted through the tracheal lumen while auscultating for breath sounds.
9. Use of the correct lumen for ventilation should be confirmed with a qualitative/quantitative end-tidal CO_2 or esophageal detector device.
10. When the device is positioned appropriately, the tube is secured with tape.

IV. VIDEO LARYNGOSCOPE

A. Indications

1. Endotracheal intubation in known or presumed difficult airway
2. Known or suspected cervical spine injury

B. Equipment

1. Bag-mask resuscitation unit with high-flow oxygen source
2. Pulse oximeter
3. Electrocardiographic monitor
4. Blood pressure monitoring
5. Gloves, mask, and eye protection
6. Video laryngoscope with appropriate blade
7. Endotracheal tube (ETT) of appropriate size for patient
8. Syringe for cuff inflation
9. Water-soluble lubricant
10. Qualitative CO_2 detector or CO_2 monitor
11. Resuscitation cart

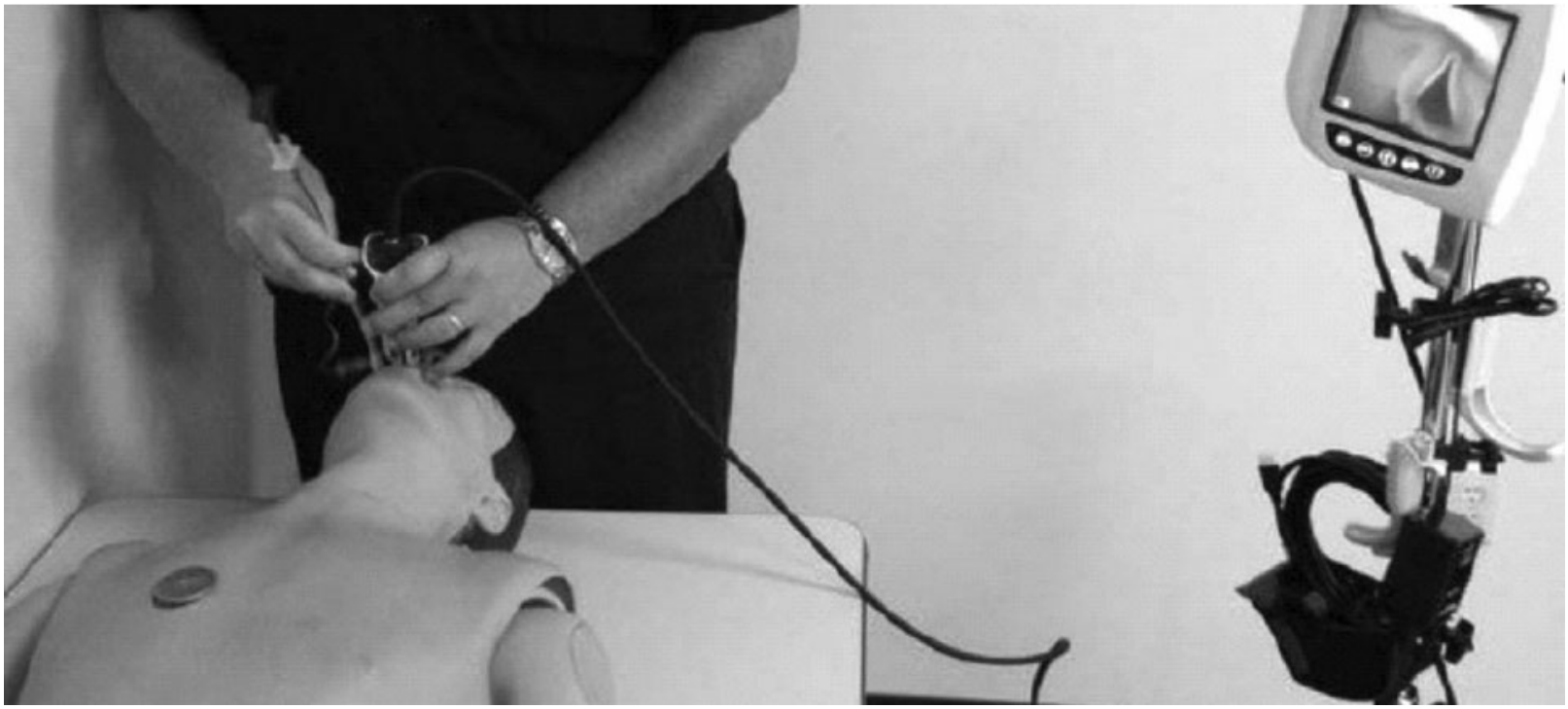
C. Preparation for insertion

1. Don gloves, mask, and eye protection.
2. Assure patent airway and optimal oxygenation and ventilation.
3. Assure IV access.
4. Apply pulse oximeter, electrocardiographic and blood pressure monitors.
5. Prepare ETT with stylet and check cuff.

6. Turn on video laryngoscope power and check light/camera.
7. Assure proper cable and blade attachment.
8. Position video screen for optimal viewing during laryngoscopy.
9. Preoxygenate with 100% oxygen for 2 to 3 minutes if time permits.

D. Technique (**Figure A5-3**)

Figure A5-3. Video Laryngoscope



Courtesy of Jason Emerson, Skaneateles Press, Eagle Newspapers, Syracuse, New York, USA.

1. The operator stands behind the head of the bed, and the bed is raised to a position of comfort for the operator.
2. The patient is placed in a neutral or sniffing position (i.e., head extended, neck flexed), unless potential or definite cervical spine injury prevents neck extension.
3. Consider lubricating the tongue side of the laryngoscope blade prior to insertion. Insert the laryngoscope blade into the oropharynx, and advance into the hypopharynx while watching the video screen for anatomical landmarks.
4. Once positioned in the hypopharynx, lift up and away, then adjust the position until the glottis and vocal cords are seen.
5. Insert and advance the ETT into the hypopharynx until the tip is seen near the end of the laryngoscope blade on the video screen.
6. While watching the video screen, advance the ETT through the glottis opening until the cuff passes the vocal cords. Make small adjustments in the laryngoscope and ETT positioning as necessary to intubate the trachea.
7. Gently remove the laryngoscope blade while holding the ETT in place. Be careful not to kink or pinch the camera cable.
8. Inflate the ETT cuff and remove the stylet. Attach the bag-valve device and provide manual ventilation. Confirm proper placement with bilateral breath sounds and a qualitative/quantitative end-tidal CO_2 or esophageal detector device.

9. When the ETT is positioned appropriately, the tube is secured with tape.

V. OPTICAL LARYNGOSCOPE

A. Indications

1. Endotracheal intubation in known or presumed difficult airway
2. Known or suspected cervical spine injury

B. Equipment

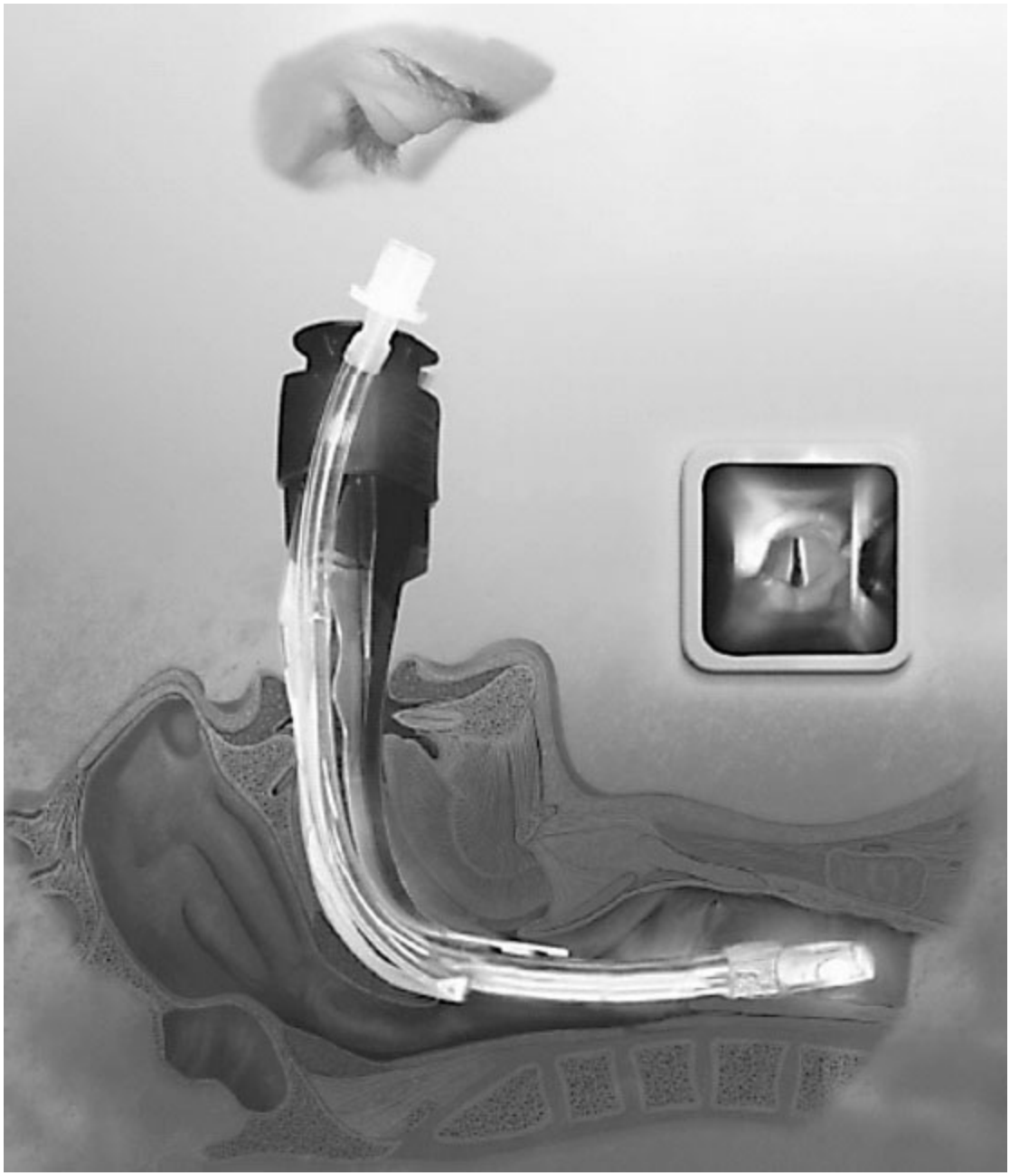
1. Bag-mask resuscitation unit with high-flow oxygen source
2. Pulse oximeter
3. Electrocardiographic monitor
4. Blood pressure monitoring
5. Gloves, mask, and eye protection
6. Appropriately sized optical laryngoscope – color coded
7. ETT of appropriate size for patient
8. Syringe for cuff inflation
9. Water-soluble lubricant
10. Qualitative CO_2 detector or CO_2 monitor
11. Resuscitation cart

C. Preparation for insertion

1. Don gloves, mask, and eye protection.
2. Assure patent airway and optimal oxygenation and ventilation.
3. Assure IV access.
4. Apply pulse oximeter, electrocardiographic and blood pressure monitors.
5. Prepare ETT, check cuff, and lubricate.
6. Choose appropriately sized optical laryngoscope.
7. Turn on laryngoscope light at least 30 seconds prior to use.
8. Load ETT into optical laryngoscope side channel.
9. Assure ETT tip is seen through eyepiece but is not obstructing view.
10. Preoxygenate with 100% oxygen for 2 to 3 minutes if time permits.

D. Technique (**Figure A5-4**)

Figure A5-4. Optical Laryngoscope



Courtesy of Prodol Meditec SA, Las Arenas Vizcaya, Spain.

1. The operator stands behind the head of the bed, and the bed is raised to a position of comfort for the operator.
2. The patient is placed in a neutral or sniffing position (i.e., head extended, neck flexed), unless potential or definite cervical spine injury prevents neck extension.
3. Consider lubricating the tongue side of the laryngoscope blade prior to insertion. Insert the laryngoscope blade in the midline over the tongue into the oropharynx and advance into the hypopharynx by rotating the laryngoscope along the tongue until it is perpendicular. Use caution not to displace the tongue posteriorly.
4. Once positioned in the hypopharynx, look through the eyepiece and lift up gently. Adjust the position until the glottis and vocal cords are seen. If glottis structures are not seen, gently pull back until seen. Do not tilt back or leverage against upper teeth or gums.
5. Advance the ETT through the glottis opening until the cuff passes the vocal cords. Make small adjustments in laryngoscope positioning as necessary to intubate the trachea.
6. Inflate the ETT cuff and separate from the laryngoscope with a gentle spreading or peeling motion. Be careful not to displace the ETT.

7. Gently remove the laryngoscope blade while holding the ETT in place. Rotate in the opposite direction from insertion.
8. Attach the bag-valve device and provide manual ventilation. Confirm bilateral breath sounds and use a qualitative/quantitative end-tidal CO_2 or esophageal detector device.
9. When the ETT is positioned appropriately, the tube is secured with tape.

Suggested Readings

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2. Danks RR, Danks B. Laryngeal mask airway: review of indications and use. *J Emerg Nurs*. 2004; 30:30-35.

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ENDOTRACHEAL INTUBATION

I. INDICATIONS

A. Loss of protective airway reflexes
B. Alteration or absence of neuronal respiratory drive
C. Increased work of breathing resulting in respiratory distress
D. Airway obstruction to gas flow
E. Alteration in patient's level of consciousness
F. Elective intubation for surgical or nonsurgical procedure
G. Hypoxic and/or hypercapnic respiratory failure

II. EQUIPMENT

A. Bag-valve-mask resuscitation unit with high-flow oxygen source – appropriate size
B. Airway mask – appropriate size
C. Yankauer and tracheal suction catheters and suction device – appropriate size
D. Monitors: blood pressure device, pulse oximeter, electrocardiography monitor
E. Laryngoscope handle and blade(s)
F. Endotracheal tubes – one size appropriate, one size smaller, and one size larger
G. Stylet – appropriate size: small, pediatric, adult
H. Oral and nasal airways – appropriate size
I. Towel roll or pad for occipital elevation
J. Medication for analgesia/anesthesia, sedation, and neuromuscular blockade
K. 10-mL syringe to inflate cuff, if appropriate
L. Qualitative CO_2 detector, CO_2 monitor, or esophageal detector device
M. Tape or tracheal tube stabilization device
N. Intravenous tubing and fluid
O. Resuscitation cart
P. Personal protective devices, gown, gloves, mask, and eye protection

III. OROTRACHEAL INTUBATION VIA DIRECT LARYNGOSCOPY

A. Preparation

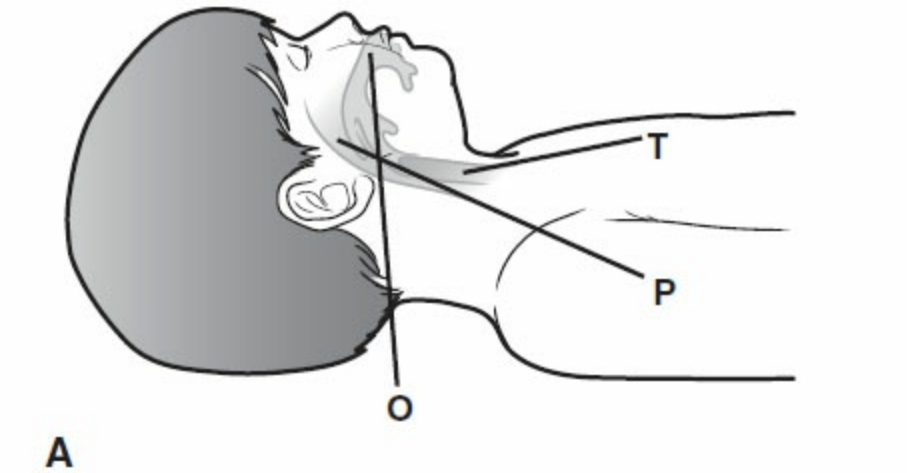
1. Assemble all equipment and ensure proper working order

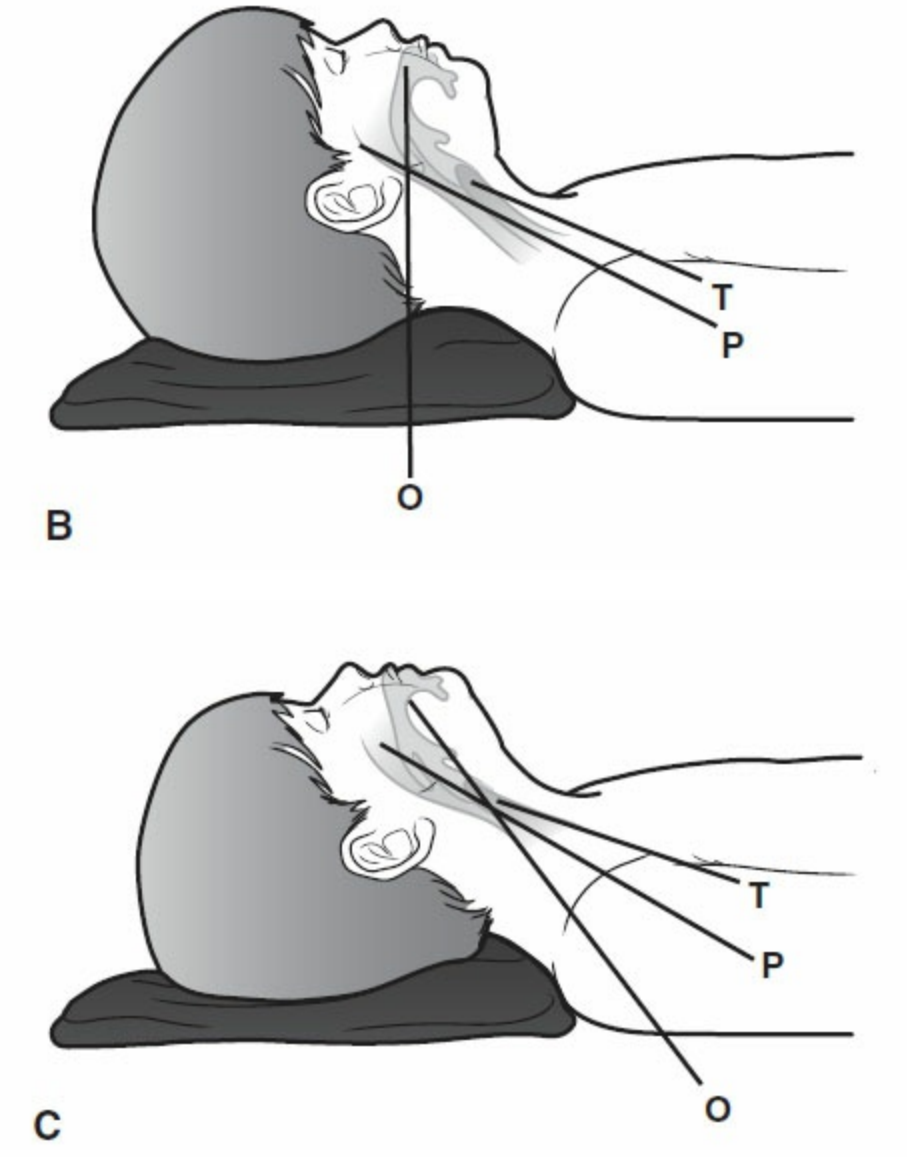
2. Don gloves, gown, mask, and eye protection
3. Confirm optimal oxygenation and ventilation
4. Establish intravenous access
5. Apply pulse oximeter, electrocardiographic, and blood pressure devices
6. Prepare the endotracheal tube
    a. If cuffed tube, check integrity by inflating and fully deflating cuff
    b. Insert stylet into endotracheal tube, bend to configuration predicted to assist glottic entry
7. Connect laryngoscope blade to handle and assure that light from bulb is bright
    a. a. Blade selection (operator's choice): larynx of the infant or small child is cephalad, appearing anterior; most clinicians do not use curved blades in infants
        i. Straight blade – used to elevate the epiglottis anteriorly
        ii. Curved blade – inserted into the vallecula
    b. Blade length: must be long enough to reach the epiglottis
8. Place the patient in the proper head position. A towel placed under the shoulders is usually needed to achieve the sniffing position in infants. Older children and adults may require a towel roll under the head to achieve the sniffing position (if cervical spine injury is not suspected).
9. Pre-oxygenate with 100% oxygen for 2-3 minutes to build up oxygen reserves, and confirm ability of operator to maintain effective ventilation
10. Proceed with analgesia and sedation; administer neuromuscular blockade only after confirmed ability to ventilate

B. Technique

1. The operator stands at the head of the patient and the bed is raised to a position of comfort for the operator. The head of the bed may be flat or raised slightly per operator preference.
2. When no cervical spine injury is suspected, place the patient in the sniffing position and gently extend the neck (**Figure A6-1**). When cervical spine injury is suspected, the neck is stabilized by an assistant and the anterior portion of the cervical collar is removed.

Figure A6-1. Positioning for Orotracheal Intubation for Children Older than 2 Years

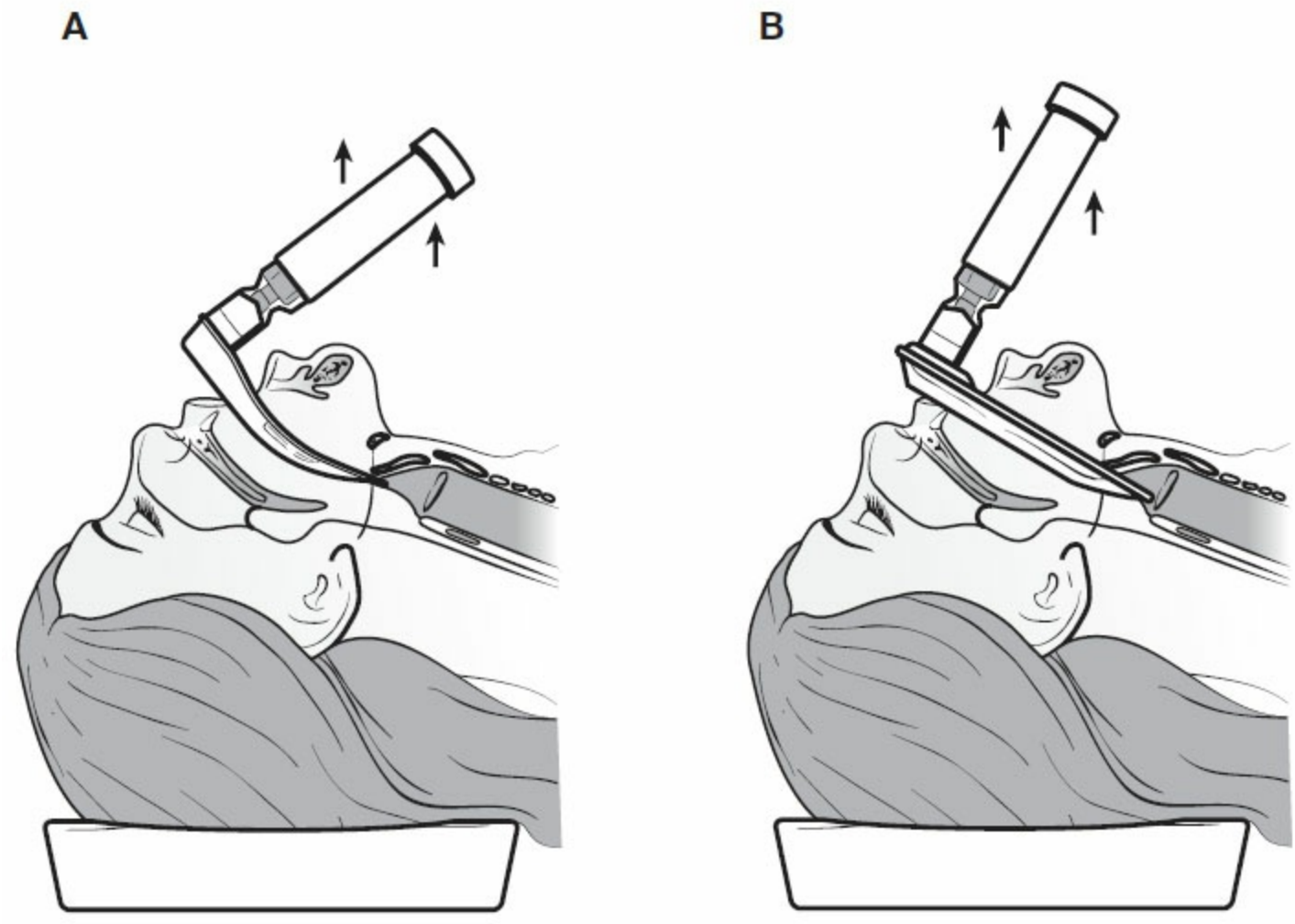




A) Maximal opening and visualization of the airway is obtained by aligning the oral (*O*), pharyngeal (*P*), and tracheal (*T*) axes. This is accomplished by placing a patient in the sniffing position. **B**) A folded sheet or towel placed under the occiput aligns the pharyngeal and tracheal axes. **C**) Extension of the neck into the sniffing position then results in approximate alignment of all three axes. Proper positioning places the external ear canal anterior to the shoulder. In children younger than 2 years, a folded towel or sheet is placed under the shoulder rather than under the occiput due to the relatively large forehead-to-occiput distance in this age group.

3. Regardless of the operator's dominant hand, the laryngoscope is always held in the left hand.
4. Cricoid pressure should be gently but firmly applied by an assistant as soon as consciousness is lost, and should be sustained until endotracheal tube placement is confirmed and the cuff is inflated, if indicated.
5. Mouth opening in the sedated/relaxed patient may be assisted by a cross-finger technique, wherein the thumb of the right hand is placed on the front lower teeth or gum of the mandible and the first finger on the upper teeth/gum of the maxilla. The mouth is gently opened by a "reverse scissor" movement of the fingers, and the laryngoscope is introduced into the mouth.
6. The tip of laryngoscope blade is inserted into the right side of the patient's mouth (**Figure A6-2**); the blade is advanced to the base of the tongue.

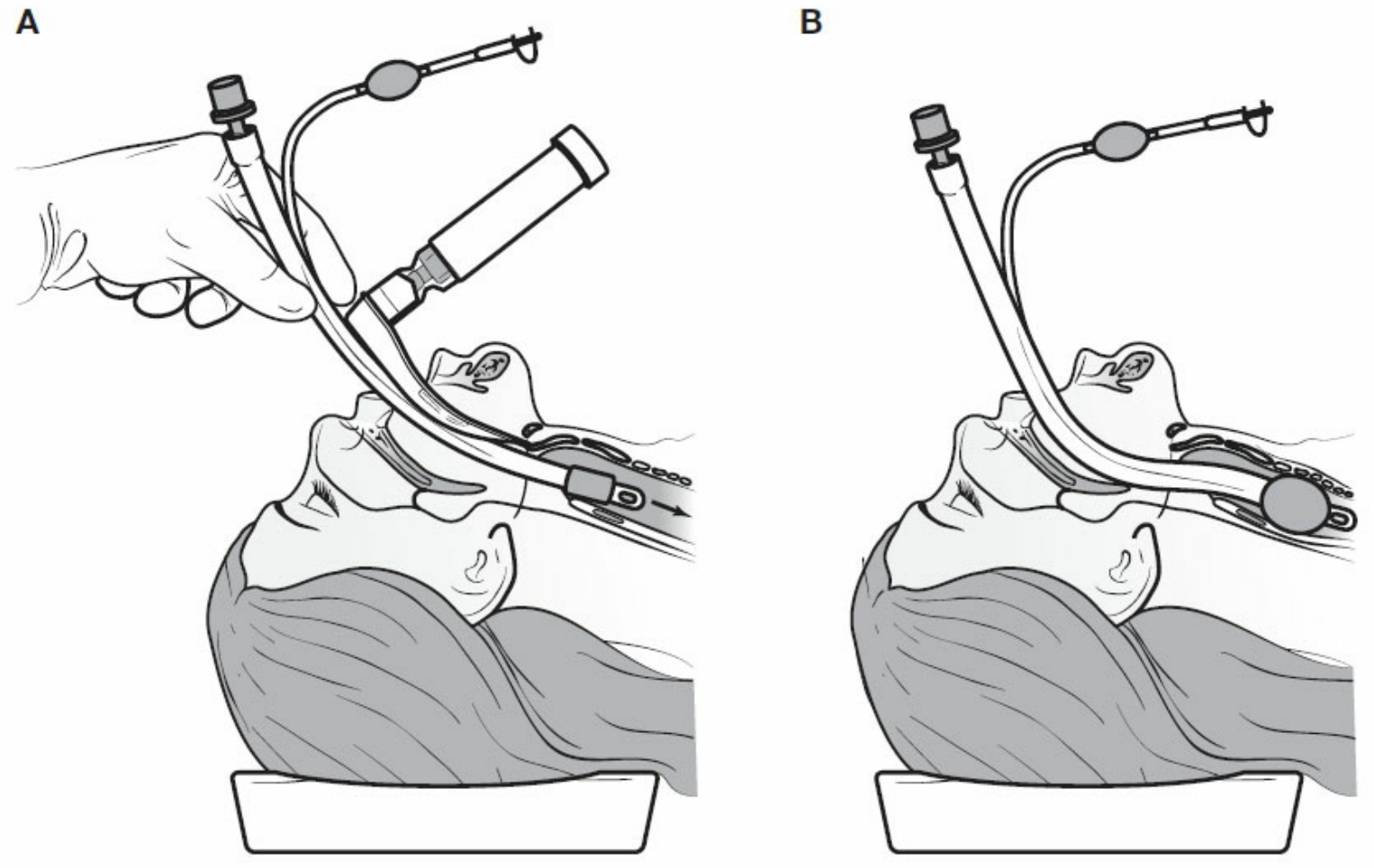
Figure A6-2. Insertion of Laryngoscope Blades



A) The curved blade follows the base of the tongue and is inserted into the vallecula. **B**) The straight blade is inserted beneath the epiglottis.

7. The tongue is swept to left; proper tongue control is key to laryngeal visualization.
8. The blade is gently advanced further to its proper position. A straight blade is placed beneath the epiglottis; a curved blade is placed into the vallecula above the epiglottis.
9. Traction should be applied only along the long axis of the laryngoscope handle as the laryngoscope lifts the tongue upward away from the larynx, revealing the glottic opening. A rocking or rotating motion of the blade and handle may damage teeth, gingiva, or lips. The base of the laryngoscope blade should never contact the upper teeth.
10. The vocal cords and glottic opening should be visualized.
11. If the vocal cords and glottis cannot be visualized, it may be helpful to have an assistant grasp the thyroid cartilage between the thumb and index finger and exert pressure until the vocal cords come into view.
12. The endotracheal tube is gently inserted through the vocal cords (**Figure A6-3**), while holding the tube/stylet with the right hand. The proper depth of insertion in centimeters can be estimated by multiplying the internal diameter of the endotracheal tube by 3 (e.g., internal diameter = 4; depth of insertion = 4 x 3 = 12 cm).

Figure A6-3. Endotracheal Tube Placement



A) The endotracheal tube is inserted through the vocal cords until the distal end rests approximately 2 to 3 cm above the carina. **B**) Once the endotracheal tube is in proper position, the laryngoscope and stylet are removed and the cuff is inflated.

13. The stylet and laryngoscope should be removed carefully. The operator must continue to hold the endotracheal tube firmly.
14. The cuff is inflated, if indicated.
15. To ensure proper position of the tube:
    a. Inspect and auscultate the chest to assure equal bilateral gas entry. In addition, auscultate the abdomen to ensure absence of air entry.
    b. Use a qualitative CO_2 detector, monitor, or esophageal detector device. Lack of color change with a qualitative CO_2 detector or low exhaled CO_2 will occur with an incorrectly placed endotracheal tube.
    c. Observe for condensation in the endotracheal tube during exhalation.
    d. Obtain chest radiograph. Tube tip should ideally be seen halfway between the thoracic inlet and carina.
16. The endotracheal tube should be secured with tape or a tube stabilization device.

IV. PEDIATRIC CONSIDERATIONS

A. Anatomic differences between adults and children

1. The larynx is more cephalad in infants than in adults, making it appear more anterior and resulting in a more difficult visualization during laryngoscopy.
2. Cricoid pressure is valuable during laryngoscopy because of the position of the larynx and assists in preventing aspiration.
3. In young children, the narrowest part of the airway is at the level of the cricoid cartilage, not at the larynx, making an anatomic "cuff" below the vocal cords.
4. In general, the diameter of the small finger approximates the proper size of the endotracheal tube. A full-term neonate can accept a tube with a 3.5-mm inner diameter.
5. When selecting a cuffed tube for a child <8 years old, the tube size should be one size smaller than the calculated size. Historically, cuffed tubes are limited to children >8 years of age (endotracheal tube size >6 mm internal diameter); uncuffed tubes are generally used in younger children. Consideration of a cuffed tube in a younger child may be necessary when a prolonged course of mechanical ventilation or high airway resistance with positive pressure ventilation is anticipated.

V. PRECAUTIONS/COMPLICATIONS

A. Hypoxia, hypercapnia during procedure
B. Cardiovascular compromise during and immediately after procedure
C. Damaged teeth, lips, gingiva
D. Malpositioned tube (right or left mainstem bronchus, esophagus)
E. Pharyngeal, laryngeal, tracheal damage
F. Gastric distension and aspiration of gastric contents
G. Bronchospasm, laryngospasm
H. Upper airway obstruction
I. Inability to visualize vocal cords
J. Inability to ventilate

Suggested Readings

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COMMON MEDICATIONS

Table A7-1	ANALGESICS AND SEDATIVES			
Agent	**Dosing**	**Onset and Duration**	**Benefits**	**Cautions**
Fentanyl	*Intermittent Dosing for Sedation/Analgesia:* 1-2 |jg/kg/dose IV Every 1-2 h	Onset: 1-2 minutes Duration: 30-60 min	-Rapid onset of action -Short acting -Relatively stable hemodynamic profile (dose dependent) -Useful for acute and chronic pain	-Chest wall rigidity syndrome (when administered rapidly [IV push]) -Respiratory depression or apnea -Lacks amnestic properties
	Continuous Sedation/Analgesia: Bolus: 1-2 |jg/kg IV over 60-90 seconds, followed by Infusion at 1-2 pg/kg/h. Titrate upward by 0.5 -1 pg/kg/h for adequate sedation/analgesia			-Narcotic abstinence syndrome and tolerance can occur with prolonged use -Reduction of gut motility/ constipation
Morphine	*Intermittent Dosing for Sedation/Analgesia:* 0.05-0.1 mg/kg/IV every 2-4 h	Onset: 5 min Duration: 2 h	-Useful for acute and chronic pain	-Respiratory depression -Histamine release -Hypotension -Pruritus -Angioneurotic edema -Nausea and emesis -Narcotic abstinence syndrome and tolerance can occur with prolonged use -Reduction of gut motility/ constipation
	Continuous Sedation/Analgesia: 0.01-0.03 mg/kg/h IV (initial infusion rate]			
Midazolam	*Intermittent Dosing for Sedation:* 0.05 to 0.1 mg/kg/dose IV every 10 min, titrate to effect to maximum of 5 mg (can be administered intranasally to calm patients without IV access]	Onset: 1-5 min Duration: 20-30 min	-Rapid onset -Short acting -Provides amnesia	-Lacks analgesic properties -Respiratory depression or apnea -Hypotension and bradycardia -Use with extreme caution or avoid altogether in setting of hemodynamic compromise

	Continuous Sedation 0.05 to 0.15 mg/kg/h IV (OR 1-2 pg/kg/min), titrate upward to achieve adequate sedation			-Withdrawal symptoms, tolerance, and myoclonus can occur with prolonged use
Lorazepam	*Intermittent Dosing for Sedation:* 0.05-0.1 mg/kg IV every 20 min, titrate to effect	Onset: 2-3 min Duration: 2-6 h	-Useful for sedation and status epilepticus -Provides amnesia	-Respiratory depression, apnea -Bradycardia -Hypotension
Ketamine	*Intermittent Dosing for Sedation:* 0.5 to 1 mg/kg/dose IV every 5 min, titrate to effect (can be administered IM to patients without IV access) *Continuous Sedation/Analgesia* 0.5-2 mg/kg/h IV [OR 5-20 Mg/kg/min] and titrate to effect	Onset: 2-3 min Duration: 15-30 mins	-Rapid onset -Airway protective reflexes remain intact -No hypotension or bradycardia (except in setting of catechol depleted state) -Excellent analgesic -Provides bronchodilation	-Increases airway secretions -Laryngospasm (blunted with atropine) -Emergence reactions (blunted with benzodiazepines)
Etomidate	*Intermittent Dosing:* 0.3 mg/kg/dose IV every 5 min, titrate to effect	Onset: 10-20 seconds Duration: 4-10 min	-Rapid onset -Short acting -Stable hemodynamic profile	-Potentially blocks stress -induced increase in cortisol causing prolonged adrenal insufficiency, potentially greater than 24 hours -Myoclonus -No analgesic properties
Thiopental	2-3 mg/kg IV, repeat as needed	Onset 30-60 seconds Duration 5-30 min	-Ultra-short-acting barbiturate -Decreases intracranial pressure -Anticonvulsant	-Cardiovascular and respiratory depression with apnea -Bronchospasm; Histamine release -Hypotension -No analgesic properties
Pentobarbital	*Intermittent Dosing:* 1-3 mg/kg IV to a maximum of 100 mg until sedated	Onset: 30-60 seconds Duration IV: 15 min	-Rapid onset IV -Useful in status epilepticus and increased intracranial pressure Can be used to induce burst suppression in elevated ICP management [3rd tier]	-Laryngospasm and respiratory depression -Can cause hypotension and bradycardia (might require pressors to maintain CPP) -No analgesic properties

	Continuous Sedation: 1-2 mg/kg/h IV, titrate upward by 1 mg/kg/h IV to achieve sedation or burst supression on EEG			-Very long half-life can lead to inability to perform a neurologic examination
Dexmedetomidine	*Intermittent Dosing:* 1 µg/kg over 10 min IV (loading dose) *Continuous Sedation:* 0.2-1.0 Mg/kg/h IV	Onset: 15 min	-Prevents delirium and shivering -Lowers requirement for benzodiazepines and narcotics -Sedative/anxiolytic without respiratory depressant effect	-Hypotension with bolus dosing -Bradycardia and heart block -Can cause PACs and PVCs -Withdrawal effects if discontinued after prolonged use (Clonidine can be used to prevent/treat withdrawal)
Choral hydrate	25-75 mg/kg PO Use lower doses in infants less than 6 months: 3mo- 30mg/kg; 4 mo- 40 mg/kg; 5 mo-5 0 mg/kg, 30 min before procedure, maximum 500 mg for infants, 1 gram for children per dose	Onset: 30-60 min Duration: 4-8 h	-Short-term sedative and hypnotic for diagnostic procedures	-May cause "hangover" effect -Paradoxical excitement can occur -Not advocated for prolonged/ repeated dosing -No analgesia

IV intravenous; EEG, electroencephalography; PO, by mouth; CPE! cerebral perfusion pressure; PAC, premature atrial contractions; PVC, premature ventricular contractions
Classified using Taketomo CK, Hodding JH, Krause DM. *Pediatric Dosage Handbook with International Trade Names Index.* 14th ed. Hudson, OH: Lexicomp; 2006-2007.

Table A7-2 NONSTEROIDAL ANTI-INFLAMMATORY DRUGS - RECOMMENDED[a,b]

Agent	Dosing	Onset and Duration	Benefits	Cautions
Acetaminophen	PO: 1-15 mg/kg every 3-4 h PR: 15-20 mg/kg every 4 h IV: 10-15 mg/kg every 4-6 h *Newborn dose:* (< 6 kg or <6 mo): PO, 10-15 mg/kg every 4-6 h PR, 20-25 mg/kg every 4-6 h	Onset: 10-60 min Duration: 1-3 h Newborn dosing: 2-5 h	-Analgesic -Antipyretic	-Maximum dose, 75 mg/kg/d -No anti-inflammatory effects
Ketorolac	IM/IV: 0.5 mg/kg every 6 h Maximum dose: 30 mg/dose	Onset: 30 min Duration: 4-6 h	-Analgesic -Anti-inflammatory agent -Antipyretic -NSAID	-Maximum 1-3 days of therapy -Avoid in renal insufficiency -Inhibits platelet aggregation and promotes bleeding -GI bleeding and ulceration can occur with prolonged use
Ibuprofen	PO: 10 mg/kg every 6-8 h	Onset: 60-120 min	-Analgesic	-Inhibits platelet

		Duration: 6-8 h	-Anti-inflammatory agent -Antipyretic -NSAID	aggregation and promotes bleeding -Should not be used with platelet count <50,000/mm^3 -Avoid in renal insufficiency -GI bleeding and ulceration can occur with prolonged use
Aspirin	PO: 10-15 mg/kg every 4 h	Onset: 15-20 min Duration: dose dependent, ranging 3-10 h with high doses (>1 g)	-Analgesic -Anti-inflammatory agent -Antipyretic -NSAID -Anti-platelet	-Contraindicated in children with low platelet counts, bleeding disorders -Associated with Reye syndrome in presence of fever and viral illness -Avoid in renal insufficiency -Avoid if peptic ulcer disease present

IV intravenous; IM, intramuscular; PO, by mouth; PR, by rectum; NSAID, nonsteroidal anti-inflammatory drug; GI, gastrointestinal

[a]These doses have a ceiling effect and maximum dose should not be exceeded.

[b]Doses given are for patients weighing <50 kg.

Classified using Taketomo CK, Hodding JH, Krause DM. *Pediatric Dosage Handbook with International Trade Names Index.* 14th ed. Hudson, OH: Lexicomp; 2006-2007.

Table A7-3 NEUROMUSCULAR BLOCKADE AGENTS*

Agent	Dosing	Onset and Duration	Benefits	Cautions
Succinylcholine (depolarizing neuromuscular blockade)	1-2 mg/kg IV to maximum dose of 150 mg	Onset: 30-60 s Duration: 4-6 min	-Rapid onset -Short duration and rapid reversal of effect	-Bradycardia leading to cardiac arrest in infants <2 months not pretreated with anticholinergic agents (atropine) -Rhabdomyolysis can occur -Causes muscle fasciculations (blunted with low-dose non depolarizing neuromuscular blocker) -Potentiates hyperkalemia (contraindicated in head trauma, crush injury, burns, hyperkalemia) -May induce neuroleptic malignant syndrome -Can trigger Malignant Hyperthermia
Vecuronium (non-depolarizing neuromuscular blockade)	*Intermittent Dosing* 0.1 to 0.2 mg/kg IV every 30 min to 1 h Induction Dose: 0.1 to 0.3 mg/kg IV higher dose	Onset: 1-3 min Duration: 30-40 min	-No fasciculations -Stable hemodynamic profile	-Slower onset -Longer duration of action -No analgesic or sedative properties Use with caution or avoid use with steroids. (Prolonged effect or

	reduces onset to 60-90 sec.			myositis may result) -Prolonged effect may occur with concomitant use of aminoglycosides
	Continuous Infusion: 0.1 mg/kg/h IV, titrate upward by 0.05 mg/kg/h to provide adequate muscle relaxation [OR 1-2 Mg/kg/min, titrate to effect]			-Provide daily NMB holiday whenever possible
Rocuronium (non-depolarizing neuromuscular blockade)	Intermittent Dosing: 0.6-1.0 mg/kg IV every 30 min to 1 h Induction dose: 0.8 to 1 mg/kg IV (Can also be administered IM if no IV access]	Onset: 30-60 s Duration: 30-40 min	-No fasciculations -Anticholinergic properties -Stable hemodynamic profile	-May cause tachycardia
	Continuous Infusion: 10-12 Mg/kg/min IV, titrate upward by 1 Mg/kg/min to provide adequate muscle relaxation			-Provide daily NMB holiday whenever possible
Cisatracurium (non-depolarizing neuromuscular blockade)	*Intermittent Dosing:* 0.1-0.2 mg/kg IV every 30-45 min	Onset: 2-3 min Duration: 35-45 min	-Can be used in renal or hepatic failure -Metabolized through Hofmann elimination	-Potential histamine release -Hypotension -Bronchospasm
	Continuous Infusion: 1-4 pg/kg/min IV, titrate upward by 0.5 pg/kg/min to provide adequate muscle relaxation			-Potentially large fluid volume as titrated up -Provide daily NMB holiday whenever possible
Atracurium (non-depolarizing neuromuscular blockade)	*Intermittent Dosing:* 0.3-0.5 mg/kg IV every 20 to 35 min	Onset: 1-4 min Duration: 20-35 min	-Can be used in renal or hepatic failure -Metabolized through Hofmann elimination	-Potential histamine release -Hypotension and bradycardia -Bronchospasm
	Continuous Infusion: 0.4 -0.8 mg/kg/h IV, titrate upward by 0.1 mg/kg/h to provide adequate muscle relaxation			-Provide daily NMB holiday whenever possible

* Should ONLY be administered in conjunction with adequate sedation and analgesia and with a controlled or secured airway and adequate ventilatory support.

NMB, neuromuscular blockade

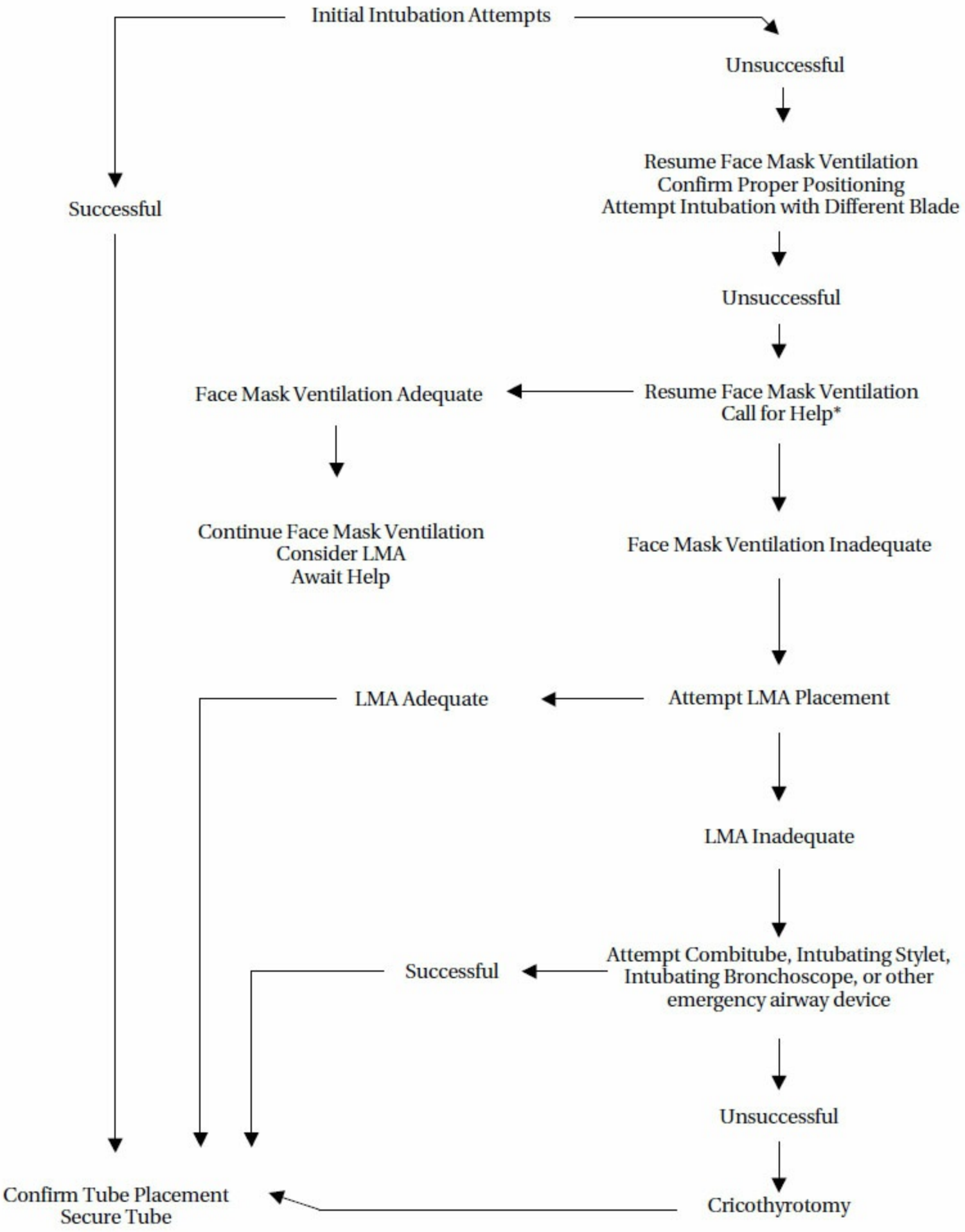
IV, intravenous; NMB, neuromuscular blockade

Classified using Taketomo CK, Hodding JH, Krause DM. *Pediatric Dosage Handbook with International Trade Names Index.* 14th ed. Hudson, OH: Lexicomp; 2006-2007.

DIFFICULT AIRWAY ALGORITHM

LMA, laryngeal mask airway
*consider contacting Anesthesia, Surgery and/or ENT services

Approach to unrecognized difficult and failed intubation.



DEFIBRILLATION AND CARDIOVERSION

I. INDICATIONS

A. Defibrillation (unsynchronized cardioversion)

1. Ventricular fibrillation
2. Pulseless ventricular tachycardia
3. Polymorphic ventricular tachycardia

B. Synchronized cardioversion

1. Unstable or stable ventricular tachycardia with a pulse
2. Unstable or stable supraventricular tachycardia, atrial fibrillation, or atrial flutter

II. EQUIPMENT

A. Conductive gel or self-adhesive defibrillation pads
B. Defibrillator/cardioverter
C. Connecting cable, leads, and electrodes
D. Medication for sedation
E. High-flow oxygen source with bag-mask oxygen delivery device
F. Emergency suction and intubation equipment
G. Pulse oximeter
H. Cardiopulmonary monitoring with electrocardiography and blood pressure monitor
I. Intravenous catheter, infusion pump, tubing, fluids
J. Resuscitation cart

III. TECHNIQUE

A. Recognize cardiac rhythm; determine severity of its physiologic effect.

B. In patients with unstable rhythms or adverse physiologic effects on systemic perfusion, begin immediate defibrillation or cardioversion after initiation of cardiopulmonary resuscitation.

C. Recognize that time delays under the above circumstances decrease the likelihood of conversion to a stable rhythm.

D. Inform and prepare patient as clinical situation dictates; sedate if necessary.

E. Assure intravenous access if time permits.

F. Provide supplemental oxygen or bag-valve-mask ventilation as indicated.

G. Monitor pulse oximeter and electrocardiography.

H. Turn on defibrillator/cardioverter.

I. Attach monitoring electrodes (if needed).

J. Apply conductive gel to paddles or apply conductive pads to chest wall.
   1. Male patients with a hirsute chest may require rapid shaving to ensure adequate contact.

K. Place paddle/electrode: Paddle placement for children is similar to that for adults.
   1. Anterolateral
      a. One paddle/electrode to right of upper sternum below clavicle
      b. One paddle/electrode to lateral of left nipple with center in midaxillary line
   2. Anteroposterior
      a. One paddle/electrode anteriorly over left precordium, below clavicle
      b. One paddle/electrode posteriorly in left infrascapular location, left of thoracic spine
   3. Avoid placement over permanent pacer or implantable cardioverter-defibrillator

L. Apply paddle pressure (if used)
   1. Adult — approximately 25 lb of pressure to each paddle
   2. Pediatric — ensure good contact with chest wall

M. Activate appropriate switch for synchronized cardioversion or unsynchronized defibrillation

N. Set energy level of electrical discharge (in accordance with recommendations of the American Heart Association)
   1. Manual defibrillator
      a. Appropriate paddle size is important; use the largest paddle size possible, assuring good chest contact over its entire area and good separation between the two paddles (about 3 cm)10
         i. Infants (<10 kg) — 4.5-cm paddles usually used
         ii. Children (>10 kg) — 8.0- to 13-cm paddles usually used

b. Be sure that the defibrillator/cardioverter provides a low-dose range for infants. Some defibrillators do not go below 10 *J* and therefore should not be used for converting infants/children weighing <20 kg (5 to 6 years of age)

c. Pediatric defibrillation
   i. 2 *J*/kg (initial)
   ii. Advance to 4 *J*/kg if rhythm persists.
   iii. Subsequent energy levels can be escalated but should not exceed 10 *J*/kg
   iv. Use adult energy levels in children ≥50 kg

d. Pediatric synchronized cardioversion
   i. Stable or unstable ventricular tachycardia: 0.5 to 1.0 *J*/kg
   ii. Stable or unstable supraventricular tachycardia: 0.5 to 1.0 *J*/kg
   iii. Advance to 2 *J*/kg if unsuccessful

e. Adult defibrillation (children >50 kg)
   i. Manual biphasic waveform device: energy level is device-specific (typically between 120 and 200 *J*). If unknown, use 200 *J* for initial shock. Subsequent shocks should be same or higher energy level as initial shock.
   ii. Monophasic waveform device: 360 *J*, initial and subsequent shocks.

f. Adult cardioversion (synchronized)
   i. Ventricular tachycardia (stable): with monophasic or biphasic device, 100 *J* for initial attempt and advance energy level as needed.
   ii. Polymorphic ventricular tachycardia: treat as ventricular fibrillation.
   iii. Atrial fibrillation: 200 *J* with monophasic device, 120 *J* to 200 *J* with biphasic device, or consult manufacturer recommendations. Escalate energy levels as needed for subsequent shocks.
   iv. Atrial flutter: 50 to 100 *J*; if rhythm persists advance energy levels as needed.
   v. Paroxysmal supraventricular tachycardia: 50 *J*; if rhythm persists, advance energy levels as needed.
   vi. If delays in synchronization occur or conditions are critical, use immediate unsynchronized shocks (defibrillation mode).

2. Automatic external defibrillator
   a. >25 kg (age 8 years and older): Use standard adult automatic external defibrillator with adult pad-cable system
   b. <25 kg (younger than 8 years): Use attenuated dose if a pediatric system is available.

Use adult system if pediatric system is not available

c. Pediatric pads may require anteroposterior placement when used on infants to avoid pad to pad contact

O. Ensure electrical safety (all personnel clear of contact with the patient, bed, and equipment).

P. Charge capacitors through defibrillator or cardioverter.

Q. Depress discharge button(s) on the device or simultaneously on the defibrillator/paddles. (With synchronized cardioversion, the discharge buttons must remain depressed until the energy is released.)

R. If defibrillation performed, immediately resume chest compressions; if cardioversion performed, assess patient (respiration, pulse, and rhythm).

S. If unsuccessful, repeat process, following standard Pediatric Advanced Cardiovascular Life Support protocols.

IV. PRECAUTIONS/COMPLICATIONS

A. During procedure

1. Skin burn may occur if insufficient gel or improper pads are used, poor contact with the chest wall occurs during discharge, or if the pads/paddles are too close to each other.
2. All metal objects should be removed from the patient to avoid skin burns.
3. Patient's environment and chest must be dry to prevent current from traveling across water, resulting in a decreased amount of delivered energy.
4. Transdermal patches should be removed as they may impede transmission of current.
5. Medical personnel may sustain electrical shock or burn if safety precautions are not followed.

B. After procedure

1. Arterial embolization
2. Pulmonary edema
3. Post-cardioversion arrhythmias; be prepared to institute cardiopulmonary resuscitation
4. Post-shock syndrome (myocardial damage)

Suggested Readings

1. American Heart Association. *Advanced Cardiovascular Life Support: Provider Manual*. Dallas, TX: American Heart Association; 2010.

2. Part 6. Electrical therapies: Automated external defibrillators, defibrillation, cardioversion, and pacing. 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122;S706-S719.

3. Wiegand D. *AACN Procedure Manual for Critical Care*. 6th ed. St. Louis, MO: Elsevier Saunders; 2010.

TEMPORARY TRANSCUTANEOUS CARDIAC PACING

I. INDICATIONS/CONTRAINDICATIONS

A. Indications

1. Symptomatic bradycardia (hypotension, chest pain, syncope, altered mental status, heart failure, etc.) unresponsive to pharmacologic management
2. Overdrive pacing of tachycardias, refractory to drug therapy or electrical cardioversion

B. Contraindications

1. Severe hypothermia
2. Not recommended for asystole

II. EQUIPMENT

A. Cardiac pacing electrode pads

B. Pulse generator

C. Connecting leads

D. Medication for sedation and/or analgesia, if necessary

E. Supplemental oxygen (cannula, mask, other as necessary)

F. Pulse oximeter

G. Electrocardiography monitor

H. Intravenous catheter, tubing, fluids

I. Resuscitation cart with pediatric and adult-sized equipment

III. TECHNIQUE

A. Recognize cardiac rhythm, determine severity.

B. Prepare patient.

C. Obtain intravenous access if not done previously.

D. Apply oxygen, monitor pulse oximetry and electrocardiography.

E. Consider pharmacologic management, including atropine, epinephrine, and/or dopamine when appropriate (follow Pediatric Advanced Cardiovascular Life Support guidelines).

F. Assemble equipment.

G. Apply electrode pads.
   1. Anteroposterior
      a. One electrode anteriorly over left precordium as close as possible to maximal cardiac impulse, below clavicle

      b. One electrode posteriorly in left infrascapular location directly behind anterior electrode, left of thoracic spine

   2. Anterolateral
      a. One electrode to right of upper sternum, below clavicle

      b. One electrode to left of nipple with center in midaxillary line

   3. Shave excessive body hair if required to ensure good contact.

H. Consider sedation and/or analgesia as necessary.

I. Connect leads to pulse generator.

J. Turn on pulse generator and monitor.

K. Set rate at 100 beats/min. Adjust as needed, based on clinical response

L. Adjust pulse generator output (mA) upward until electrical and mechanical ventricular capture (threshold) occurs (usually 20 to 60 mA). Set output 2 mA above threshold to allow for safety margin. In the setting of severe symptoms or bradycardia, it may be appropriate to start at the maximal output and then decrease if capture is achieved.

M. Be mindful of criteria for proper electrical capture.
   1. Pacer spike followed by a ventricular complex 100% of the time

   2. Wide QRS complex

   3. T wave in an opposite deflection from baseline as the QRS complex

N. Assess efficacy of mechanical capture; obtain blood pressure and palpate pulse distal to carotid site as electrical muscle stimulation from the pacemaker may mimic a carotid pulse.

O. Arrange for temporary or permanent transvenous pacemaker as necessary.

IV. PEDIATRIC CONSIDERATIONS

A. Bradycardia in children is most often secondary to hypoxemia.

B. Pacing for bradycardic rhythms secondary to hypoxemic insult may be considered after airway management, oxygenation, ventilation, chest compressions, epinephrine bolus (0.01 mg/kg, 1:10,000 concentration) and epinephrine infusion, and possibly atropine bolus (0.02 mg/kg, may repeat; minimum dose 0.1 mg and maximum total dose 1 mg) have been accomplished.

C. The effectiveness of cardiac pacing in this setting is unproven.

D. Even if electrical capture of the heart is accomplished, contractility and myocardial blood flow may not improve without mechanical capture.

E. It is recommended to use the largest available paddles or self-adhering electrode pads that will fit on the chest wall without touching (allow at least 3 cm between paddles or pads).
   1. For children >10 kg (>1 year of age), use large adult paddles.
   2. For children <10 kg (<1 year of age), use small infant paddles (4.5 cm).

V. PRECAUTIONS AND COMPLICATIONS

A. Inability to capture (~20% of patients), usually related to delay in attempting to pace

B. Painful skeletal muscle contraction

C. Skin or tissue damage

D. Temporizing measure only, before transvenous pacing

Suggested Readings

1. American Heart Association. *Advanced Cardiovascular Life Support: Provider Manual*. Dallas, TX: American Heart Association; 2011.
2. Link MS, Atkins DL, Passman RS, et al. Part 6: electrical therapies: automated external defibrillators, defibrillation, cardioversion, and pacing: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(18 Suppl 3):S706-S719.
3. Wiegand D. *AACN Procedure Manual for Critical Care*. 6th ed. St. Louis, MO: Elsevier Saunders; 2010.

THORACOSTOMY

I. INDICATIONS AND CONTRAINDICATIONS

A. Indications

1. Tension pneumothorax
2. Large simple pneumothorax
3. Penetrating thoracic wound with concurrent need for positive-pressure ventilation
4. Hemothorax
5. Symptomatic pleural effusion (recurrent, following thoracentesis)
6. Empyema
7. Chylothorax

B. Contraindications

1. Coagulopathy
   a. Condition must be corrected before nonemergent thoracostomy.
   b. Risk of hemorrhage must be accepted with tension pneumothorax.
2. Inability to aspirate fluid or air to confirm a patent pleural space
   a. This dictum holds in all circumstances except a penetrating thoracic wound with need for positive-pressure ventilation.
   b. Attempted tube placement in the presence of an obliterated pleural space risks pulmonary injury and potentially fatal hemorrhage.
   c. Aspiration is performed most conveniently through the thoracostomy incision immediately before tube placement.
   d. Aspiration is most important when an apparent effusion presents as a “whiteout” on a chest radiograph, and its free-flowing nature cannot be confirmed radiographically. Such an apparent effusion may be solid tumor, and blunt dissection into such tumor could have devastating hemorrhagic consequences.

II. EQUIPMENT

A. Needle thoracostomy

1. 14- to 16-gauge catheter over needle
2. 23-gauge butterfly needle (infants)

B. Surgical tube thoracostomy

1. Sterile gloves, gown, eye protection, mask, cap, and drapes
2. Intravenous catheter, tubing, and fluid
3. Supplemental oxygen
4. Monitors (electrocardiographic, pulse oximeter)
5. Skin disinfectant
6. Sterile syringes and infiltrating needles
7. Local anesthetic
8. Scalpel with #10 or #15 blade
9. Forceps
10. Curved clamp
11. 12F to 40F thoracostomy tube
    a. Approximate sizes for pediatric thoracostomy tubes by age and weight are listed in **Table A11-1**.

Table A11-1 APPROXIMATE SIZES FOR PEDIATRIC THORACOSTOMY TUBES BY AGE AND WEIGHT[a]

Age	Approximate Weight (kg)	Tube Size (French)
Newborn to 9 months	3.5-8	12-18
10 to 17 months	10	14-20
18 months to 3 years	12-15	14-24
4 to 7 years	17-22	20-32
8 years	28	28-32
≥9 years	≥35	28-38

[a]Needle thoracostomy can usually be accomplished on infants with a 23-gauge butterfly needle.

b. 32F to 40F thoracostomy tubes are placed in trauma settings to evacuate an acute hemothorax that potentially contains clots. The largest diameter tube accommodated by the intercostal space is used in this circumstance.

12. If placement of the chest tube is nonemergent and an experienced technician is available, thoracic ultrasound can be a useful adjunct to help localize lung pathology (pleural fluid, pneumothorax) and guide correct positioning of the chest tube.
13. Water-seal drainage system
14. Needle holder
15. 0-silk or 0-polypropylene suture on cutting needle
16. Suture scissors
17. 1/4-inch-wide adhesive tape strips or cable ties with applicator
18. Sterile 4 × 4 gauze sponges
19. Antiseptic ointment
20. 4-inch-wide impervious tape strips
21. 1-inch-wide adhesive tape
22. Resuscitation cart

C. Seldinger technique chest tube

1. Sterile gloves, gown, eye protection, mask, cap, and drapes
2. Intravenous catheter, tubing, and fluid
3. Supplemental oxygen
4. Monitors (electrocardiographic, pulse oximeter)
5. Skin disinfectant
6. Sterile syringes and infiltrating needles
7. Local anesthetic
8. Appropriately sized thoracostomy tube with dilator (**Table A11-1**)
9. Seldinger guidewire
10. Scalpel with #10 or #15 blade
11. Water-seal drainage system
12. Sterile 4 × 4 gauze sponges

13. Antiseptic ointment
14. 4-inch-wide impervious tape strips
15. 1-inch-wide adhesive tape
16. Resuscitation cart
17. If placement of the chest tube is nonemergent and an experienced technician is available, thoracic ultrasound can be a useful adjunct to help localize lung pathology (pleural fluid, pneumothorax) and guide correct positioning of the chest tube.

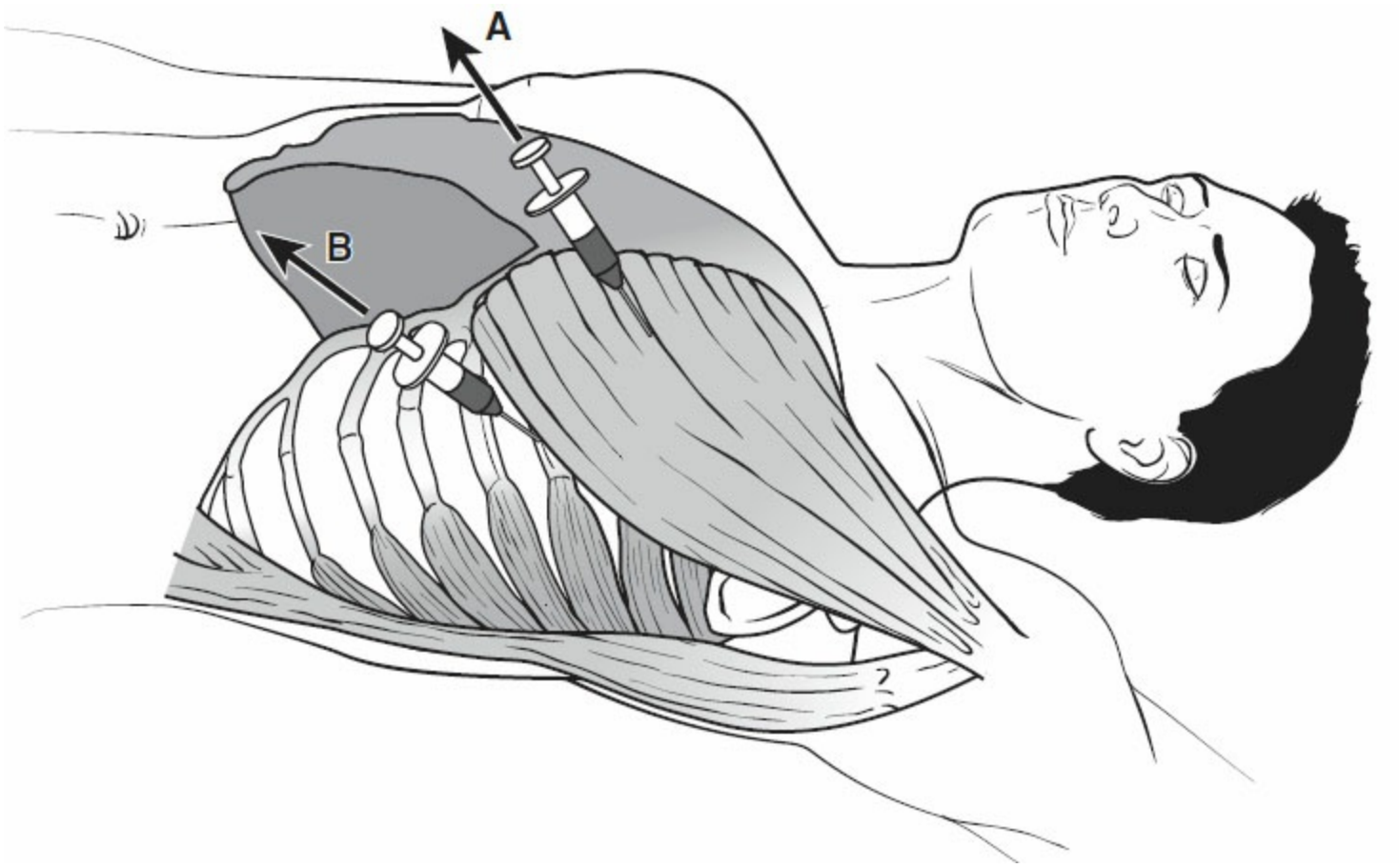
III. TECHNIQUE: SURGICAL CHEST TUBE PLACEMENT

A. Analgesia/sedation

1. Surgical tube thoracostomy is a painful procedure. In nonemergent and semi-urgent circumstances, intravenous narcotic analgesia and a benzodiazepine should be titrated to effect as hemodynamic and respiratory status allow. Local anesthetic should be infiltrated generously throughout the tube thoracostomy tract.

B. Preliminary needle thoracostomy (**Figure A11-1**)

Figure A11-1. Sites for Needle Thoracostomy



A, Second intercostal space, midclavicular line. **B**, Fifth intercostal space, midaxillary line. The latter is also the incision site

for placement of a thoracostomy tube and necessitates transgression of much less chest wall musculature and no breast tissue.

1. 1. Perform before tube thoracostomy for rapid temporizing treatment of tension pneumothorax.
2. 2. Select site.
    a. Midclavicular line at the second intercostal space; necessitates that the pectoralis major muscle and possibly breast tissue be penetrated before the intercostal space.

    b. Midaxillary line at the fifth intercostal space; placed in the auscultatory triangle posterior to the pectoralis and anterior to latissimus dorsi muscles where only the thin serratus anterior muscle need be penetrated before the intercostal space.
3. Don cap, mask, eye protection, and sterile gloves.
4. Quickly prepare the access site with povidone-iodine solution for children younger than 2 months; if older than 2 months, cleanse site with chlorhexidine solution.
5. Advance 14-gauge catheter over needle with attached syringe immediately over the superior aspect of the rib while aspirating.
6. When air is aspirated, advance catheter completely, and withdraw needle and syringe. Withdraw catheter following completion of tube thoracostomy.
7. Note: 1 mL of saline in the aspirating syringe reveals access of intrapleural air, recognized as bubbles.

C. Preparation for surgical tube thoracostomy

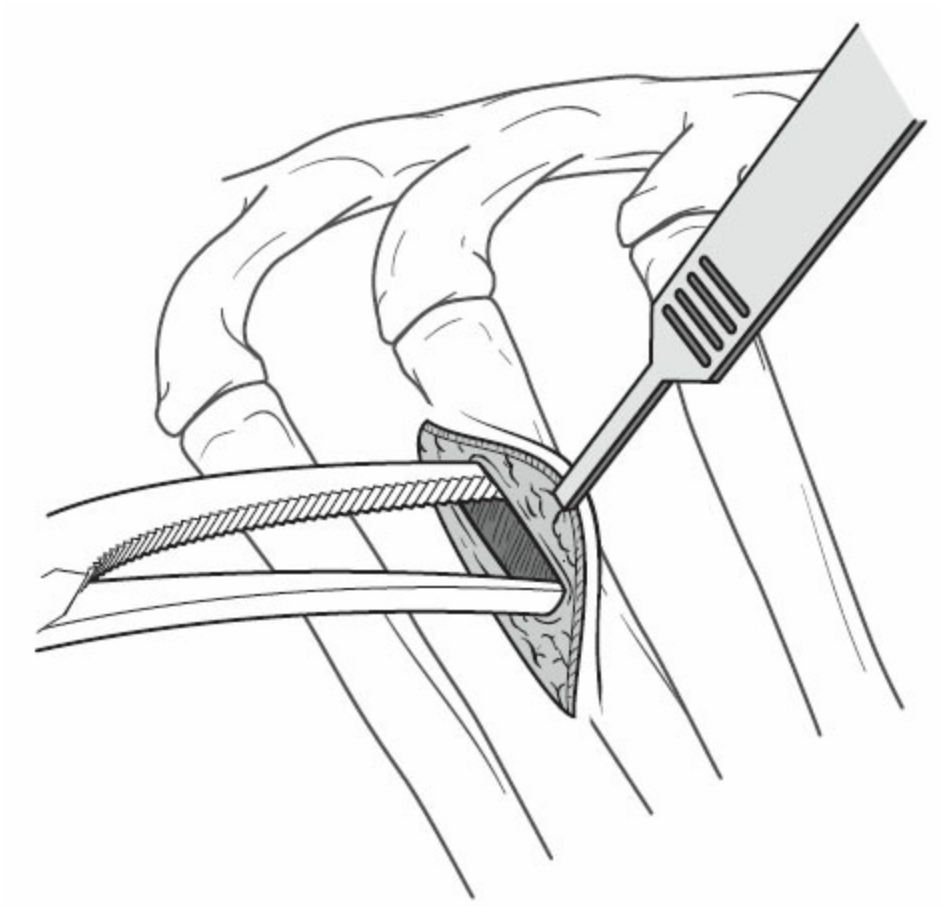
1. Provide supplemental oxygen.
2. In nonemergent circumstances, establish intravenous access, electrocardiographic monitoring, and pulse oximetry.
3. Assemble the following nonsterile materials: water-seal drainage system, 1/4-inch-wide strips of adhesive tape or cable ties to secure thoracostomy tube to drainage system, 4-inch-wide impervious tape strips to secure dressing.
4. Ensure adequate lighting.
5. Place patient in supine position with ipsilateral arm extended.
6. Don cap, mask, eye protection, and sterile gloves.
7. Cleanse patient's anterior and lateral chest wall with antiseptic solution. Remove gloves.
8. Don sterile gown and gloves.
9. On a sterile work space, lay out, from left to right, the following sterile instruments and materials in sequence: syringe with infiltrating needle loaded with local anesthetic, scalpel with blade, forceps, curved clamp, thoracostomy tube, needle holder loaded with suture, suture

scissor, dressing composed of gauze 4 × 4 sponges, and antiseptic ointment. These instruments and materials will be used in this sequence.

D. Insertion

Instrument	Maneuver
1. Syringe and needle with local anesthetic	a. Raise the cutaneous wheal at the incision site. b. Deeply infiltrate the underlying subcutaneous tissue.
2. Scalpel	a. Make a 3-cm incision at position *B* in **Figure A11-1**, through the skin and subcutaneous tissue of the fifth intercostal space. b. Deepen the incision to the level of the chest wall musculature. *Note*: The skin incision parallels the intercostal space. It should be placed one interspace below the intended level of pleural entry so that a tract deep to subcutaneous tissue can be created for the tube. This tract closes spontaneously upon tube removal.
3. Syringe and needle with local anesthetic	a. Through the wound, infiltrate subcutaneous tissue cephalad to the incision.
4. Forceps	a. Using the left hand, retract subcutaneous tissue cephalad away from chest wall to create tension at the junction of subcutaneous tissue and chest wall musculature (**Figure A11-2**).

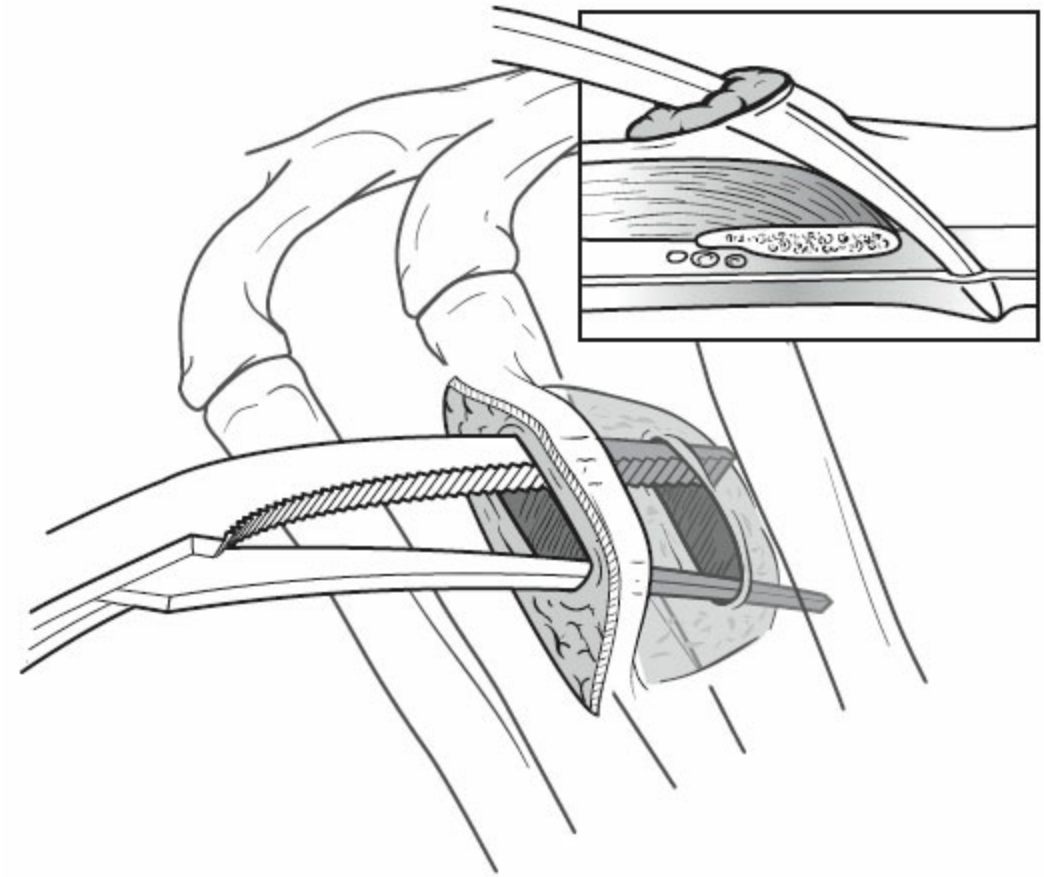
Figure A11-2. Blunt Dissection for Thoracostomy



Retraction of skin and subcutaneous tissue with blunt dissection of subcutaneous tissue from chest wall musculature superior to incision.

5. Curved clamp	a. Continue to apply above traction with forceps. b. With curved clamp in the right hand, spread at junction of subcutaneous tissue and chest wall musculature to open this plane (**Figure A11-2**).
6. Syringe and needle with local anesthetic	a. Through the wound, infiltrate musculature and pleura of the fourth intercostal space. b. Advance the needle into the pleural space while aspirating syringe. c. Confirm the presence of air or fluid in the pleural space.
7. Curved clamp	a. With curved clamp in the right hand, hold tips against the superior aspect of the fifth rib with concavity of the clamp facing the pleural space. b. Perform intercostal dissection immediately superior to a rib to avoid injury to the neurovascular bundle that lies inferior to each rib. c. Advance curved clamp through musculature (serratus anterior and intercostal muscles) and pleura into the pleural space (**Figure A11-3**). *Note*: This maneuver may need to be forceful but must always be controlled. A dramatic loss of resistance will signal entry into the pleural space and will be followed by an egress of fluid and/or air. d. Ensuring that the tips of the clamp remain on the superior aspect of the fifth rib, widely separate the jaws of the clamp to create a generous opening through the serratus anterior muscle, intercostal muscles, and pleura. e. Insert the left index finger into the pleural space as the clamp is withdrawn. Palpation of the smooth pleura confirms the intrapleural location. Sweep the finger through 360° to ensure the absence of adhesions between parietal and visceral pleura. Such adhesions and obliteration of the pleural space would predispose the patient to pulmonary injury during tube insertion.

Figure A11-3. Creation of Pleural Opening

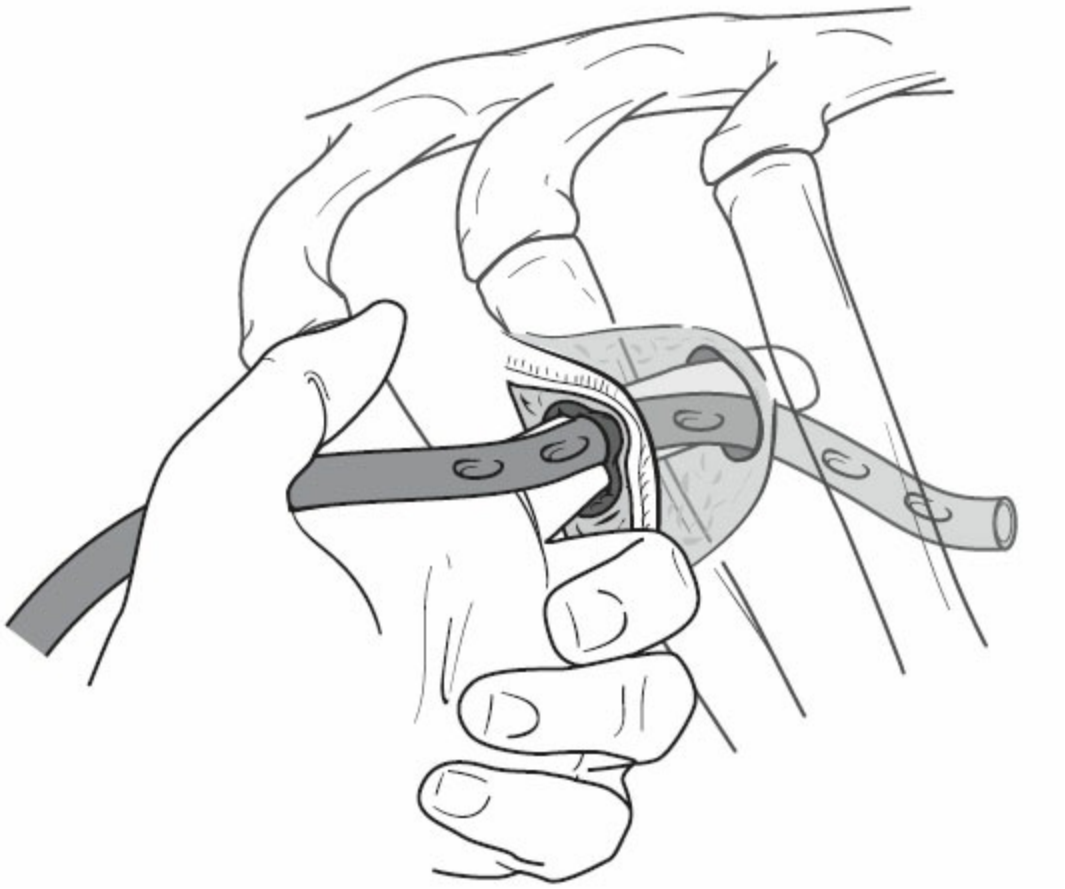


The curved clamp has been advanced into the superior aspect of the incision and advanced through the intercostal musculature and pleura at the superior margin of the fifth rib. The jaws of the clamp are then spread to create a pleural opening. The opening should be generous enough to simultaneously admit a finger and the thoracostomy tube. Insert shows the path of the curved clamp in cross section.

8. Thoracostomy tube

a. Keep the left index finger in the pleural space.
b. With the right hand, advance the thoracostomy tube over the tip of the left index finger into the pleural space (**Figure A11-4**). Passage of the tube over the tip of the intrapleural index finger ensures intrapleural placement of the tube.
c. Advance the tube until resistance is encountered (approximately 15 to 25 cm). The last side hole of the tube should reside 2 cm within the pleural cavity. Ideally, the tip of the tube lies at the pleural apex.

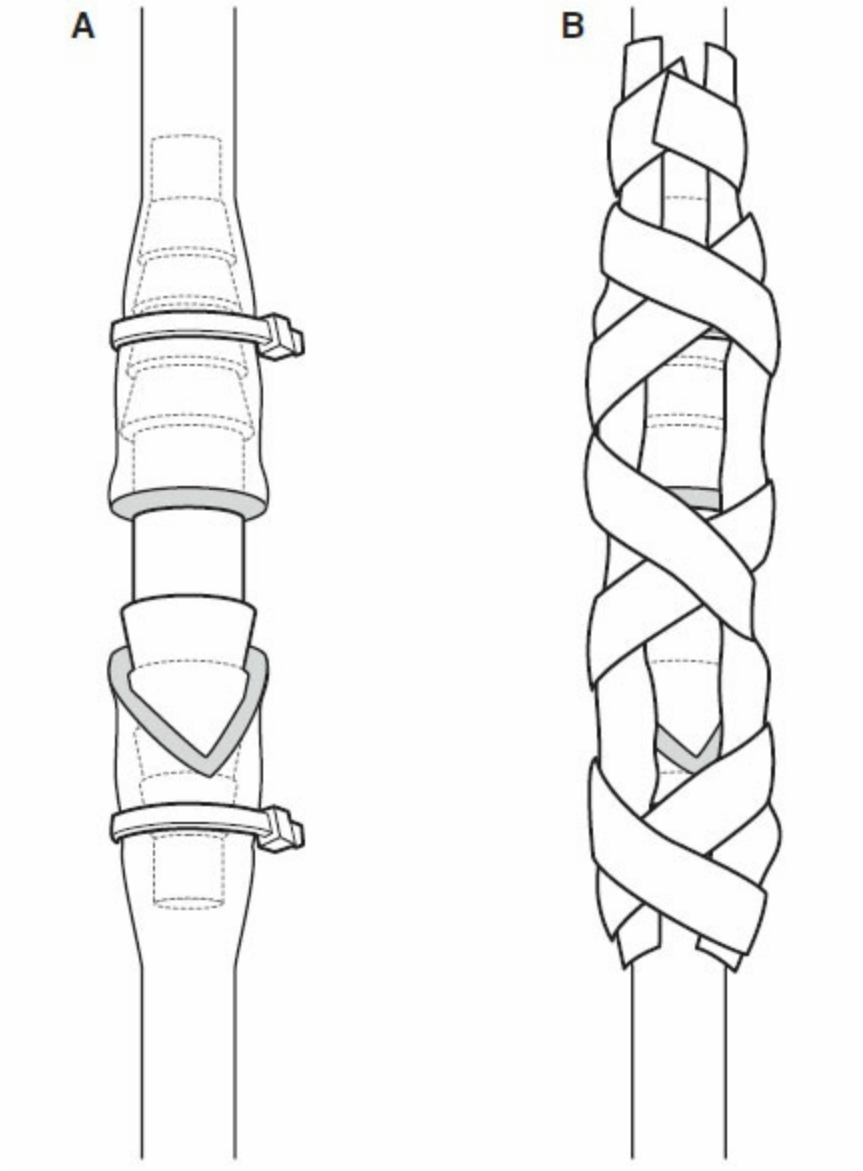
Figure A11-4. Placement of Thoracostomy Tube



The left index finger replaces the curved clamp and remains within the pleural space as the thoracostomy tube is advanced over the tip of the finger, ensuring intrapleural location.

9. Water-seal drainage system	a. Connect the thoracostomy tube to the drainage system.
10. Needle holder and suture	a. Place a suture of 0-nonabsorbable material through the wound on either side of the thoracostomy tube. b. Tie each suture to close the wound. c. Tie each suture about the thoracostomy tube to secure it. d. Place additional sutures as necessary to close the wound.
11. 1/4-inch adhesive tape	a. Secure the connection between chest tube and drainage or cable tie system tubing (**Figure A11-5**). b. Do not allow tape to obscure the connection. One must be able to see that the connections are intact at all times.

Figure A11-5. Connection of Thoracostomy Tube



The thoracostomy tube and tubing from the draining system are secured about a conical connecting adaptor with cable ties (A) or strips of adhesive tape placed longitudinally and in a spiral fashion (B).

12. Gauze dressing, 4 x 4 sponges, antiseptic ointment, and impervious tape	a. Place dressing over the thoracostomy site. b. Secure the dressing with impervious tape.
13. 1-inch adhesive tape	a. Secure chest tube and drainage system tubing to patient's trunk.

IV. TECHNIQUE: NONSURGICAL CHEST TUBE PLACEMENT (SELDINGER TECHNIQUE)

A. Analgesia/sedation

1. Nonsurgical chest tubes do not require chest wall dissection to reach the pleural cavity.
2. Consider moderate to deep sedation

3. Local chest wall anesthetic minimizes the need for deeper level of sedation.

B. Preliminary needle thoracostomy (**Figure A11-1**)

1. 1. Perform as in **Section IIIB**.

C. Preparation for nonsurgical tube thoracostomy

1. Provide supplemental oxygen.
2. In nonemergent circumstances, establish intravenous access, electrocardiographic monitoring and pulse oximetry.
3. Assemble the following nonsterile materials: water-seal drainage system, 1/4-inch-wide strips of adhesive tape or cable ties to secure thoracostomy tube to drainage system, 4-inch-wide impervious tape strips to secure dressing.
4. Ensure adequate lighting.
5. Place patient in supine position with ipsilateral arm abducted.
6. Don cap, mask, eye protection, and sterile gloves.
7. Cleanse patient's anterior and lateral chest wall with antiseptic solution. Remove gloves.
8. Don sterile gown and gloves.
9. On a sterile work space, lay out, from left to right, the following sterile instruments and materials in sequence: syringe with infiltrating needle loaded with local anesthetic, scalpel, 18-gauge needle, thoracostomy tube with dilators inside, Seldinger wire, needle holder loaded with suture, suture scissors, dressing composed of gauze 4 × 4 sponges, and antiseptic ointment. These instruments and materials will be used in this sequence.

D. Insertion

Instrument	Maneuver
1. Syringe and needle with local anesthetic	a. Raise cutaneous wheal at the incision site. b. Deeply infiltrate the underlying subcutaneous tissue and attempt to reach pleural fluid or air.
2. 18-gauge needle and syringe	a. Along the numbed tract, insert an 18-gauge introducer needle over the superior aspect of the rib into the pleural space. Once the pleural cavity is reached, aspirate fluid or air and disconnect needle syringe, making sure not to allow atmospheric air inside needle.
3. Scalpel	a. Make the 1-cm superficial skin incision at entrance to numbed tract to allow the dilator and thoracostomy tube to fit into the pleural space.

4. Seldinger wire	a. Advance wire through a hollow-bore 18-gauge needle until the wire is inside the pleural cavity space, making sure to advance the wire only 2 to 3 cm beyond the length of the needle. Withdraw the needle and place it in a safe location on the tray.
5. Thoracostomy	a. Advance thoracostomy tube and smallest dilator over wire until resistance tube and dilators is felt, then lightly push through the resistance (entering pleural space). Continue to dilate the tract and opening in the pleural space by advancing in sequence (small to large) the supplied dilators over the wire. Advance the chest tube off the dilator until confident that the chest tube holes are in the pleural cavity, then withdraw the dilator.
6. Water-seal drainage system	a. Connect the thoracostomy tube to the drainage system.
7. Needle holder and suture	a. Place a suture of 0-nonabsorbable material through the wound on and suture either side of the thoracostomy tube. b. Tie each suture to close the wound. c. Tie each suture about the thoracostomy tube to secure it. d. Place additional sutures as necessary to close the wound.
8. 1/4-inch adhesive tape or cable ties	a. Secure connection between the chest tube and drainage system tubing (**Figure A11-5**). b. Do not allow tape to obscure the connection from view. One must be able to see that the connections are intact at all times.
9. Gauze dressing, 4 x 4 sponges, antiseptic ointment, and impervious tape	a. Place dressing over the thoracostomy site. b. Secure dressing with impervious tape.
10. 1-inch adhesive tape	a. Secure the chest tube and drainage system tubing to the patient's trunk.

E. Pleural decompression

1. Adjust suction to -20 cm H_2O.
2. Consider prophylactic antibiotic coverage.

F. Thoracostomy tube monitoring

1. Monitor the thoracostomy tube with portable chest radiography to ensure appropriate tube placement and the absence of iatrogenic pneumothorax. The last side hole of the thoracostomy tube lies on a radiopaque line and thus is visible on the radiograph as a gap in this line; the gap should always appear well within the pleural space.
2. Confirm chest tube patency by the presence of a to-and-fro movement of fluid with respiration

(respiratory variation). This may be detected within the thoracostomy tube, the tubing of the collection device, or the water-seal chamber. As the pleural space is definitively decompressed, the thoracostomy tube will become loculated from the general pleural space by visceral and parietal pleural adhesion; respiratory variation will then be lost.

3. Assess the character and volume of pleural drainage frequently. The significance of diminished drainage volume can only be determined by concurrent chest radiographic findings. For example, diminishing sanguineous drainage may mean cessation of bleeding or occlusion of the thoracostomy tube by clot; the chest radiograph will reveal increasing effusion/hemothorax in the latter circumstance, but not the former.

4. Check for air leaks, apparent as air bubbling through the water seal (not the suction regulator). Small air leaks will demonstrate bubbling only during spontaneous expiration or mechanical inspiration. Large air leaks will demonstrate bubbling through both phases of the respiratory cycle. These continuous air leaks may indicate a bronchopleural fistula or tracheobronchial laceration.

G. Thoracostomy tube removal

1. General criteria for thoracostomy tube removal
   a. Complete radiographic expansion of the lung
   b. Absence of air leak for 24 hours
   c. Drainage volume <100 mL over 24 hours
2. Prepare a dressing of impervious tape, gauze 4 × 4 sponges, and antiseptic ointment.
3. With sterile scissors, divide the sutures securing the thoracostomy tube.
4. Instruct the patient to take a full inspiration, hold the breath, and perform a Valsalva maneuver. Practice this sequence several times.
5. Repeat the sequence, briskly withdrawing the thoracostomy tube at full inspiration of the Valsalva maneuver, and immediately apply the occlusive dressing to the thoracostomy wound.
6. Do not close the thoracostomy site with suture or other material.
7. Obtain an immediate portable chest radiograph to ensure the absence of pneumothorax.

V. PRECAUTIONS AND COMPLICATIONS

A. Possible injury to intercostal artery, vein, or nerve

B. Extrapleural tube position, including subdiaphragmatic placement

C. Subcutaneous emphysema

D. Break in water seal, resulting in pneumothorax

E. Chest wall hematoma/ecchymosis

F. Chest wall or intrapleural hemorrhage

G. Infection
   1. Insertion-site cellulitis
   2. Tract infection
   3. Empyema

H. Laceration of diaphragm or intrathoracic/intra-abdominal viscera

I. Recurrence of pneumothorax (upon removal, secondary to entrained room air or rupture of pulmonary bulla/bleb)

J. Life-threatening tension pneumothorax if chest tube is clamped in the presence of an air leak

Suggested Readings

1. Etoch SW, Bar-Natan MF, Miller FB, Richardson JD. Tube thoracostomy. Factors related to complications. *Arch Surg*. 1995;130:521-526.
2. Havelock T, Teoh R, Laws D, et al. Pleural procedures and thoracic ultrasound: British Thoracic Society Pleural Disease Guideline 2010. *Thorax*. 2010;65:ii61-ii76.
3. Light RW. Pleural controversy: Optimal chest tube size for drainage. *Respirology*. 2011;16:244-248.
4. Lotano VE. Chest tube thoracostomy. In: Parrillo JE, Dellinger RP, eds. *Critical Care Medicine*. 3rd ed. St. Louis, MO: Mosby, Inc., 2008; 271-279.
5. Martino K, Merrit S, Boyakye K, et al. Prospective randomized trial of thoracostomy removal algorithms. *J Trauma*. 1999;46:369-373.
6. Richardson JD, Spain DA. Injury to the lung and pleura: In: Mattox KL, Feliciano DV, Moore EE, eds. *Trauma*. 4th ed. New York, NY: McGraw-Hill, 2000; 523-543.

CENTRAL VENOUS ACCESS

I. INDICATIONS

A. Poor peripheral venous access

B. Administration of multiple incompatible medications

C. Administration of vasoactive, irritating, caustic, or hypertonic solutions

D. Hemodynamic monitoring

E. Insertion of a transvenous cardiac pacing catheter

F. Support of rapid infusion of resuscitation fluids

G. Support of frequent blood sampling; minimizing repeated venipuncture

H. Support of hemodialysis

II. EQUIPMENT

A. Sterile central venous catheter (antibiotic-impregnated catheter is recommended); guidewire; 18-, 22-, or 25-gauge needle, according to catheter size; or central venous catheter kit (singlelumen or multilumen central venous catheter)

B. Consider use of ultrasound for central or peripheral insertion of central catheter: reduces number of attempts, increases rate of success

C. Consider use of peripherally inserted central catheter: may be appropriate for poor or difficult venous access, irritating or hypertonic solutions, or frequent blood sampling

D. Syringe, scalpel, dilator, suture or securement device, infiltrating needles

E. Saline or heparinized saline solution

F. Semitransparent dressing

G. Chlorhexidine-impregnated patch for patients ≥2 months old, per institution-specific protocol

H. Sterile 2x2 and 4x4 gauze sponges

I. Medication for local anesthesia and sedation

J. Sterile gloves, gown, mask, protective eye gear, hat, drapes

K. Chlorhexidine for children ≥ 2 months old, povidone-iodine for infants <2 months old, or per institution policy

L. Supplemental oxygen

M. Pulse oximeter

N. Electrocardiographic monitor

O. Intravenous tubing and fluid

P. Resuscitation cart available

III. TECHNIQUE: MODIFIED SELDINGER

A. Obtain consent.

B. Inform the patient using appropriate developmental approach.

C. Notify licensed provider for appropriate timing of sedation.

D. Choose catheter size and site, gather equipment.

E. Ensure appropriate cardiorespiratory monitoring.

F. Conduct final patient verification process.

G. Assure patent peripheral intravenous or intraosseous access, if possible.

H. Assess anatomy of proposed central venous cannulation site with ultrasound, if available.

I. Put on hat, mask, and protective eye gear.

J. Wash hands.

K. Position the patient and identify anatomical landmarks (**Table A12-1**).

Table A12-1 INSERTION SITE AND PATIENT POSITIONING

Internal Jugular Site	**Subclavian Site**	**Femoral Site**
Patient position: Trendelenburg	Patient position: Trendelenburg	Patient position: Supine with hip Abduction and leg in external rotation on side of cannulation
1. Turn patient's head to side opposite of insertion site.	1. Turn patient's head to side of insertion site.	Place roll under hip side to be cannulated.
2. Extend neck and head by placing a towel under the shoulder on side of insertion site if no cervical spine injury	2. Place shoulder roll under and parallel along the spine and hyperextend the neck, if no cervical	

is present.	spine injury is present.	
Internal Jugular Site Landmark	**Subclavian Site Landmark**	**Femoral Site Landmark**
1. Medial and lateral bellies of the sternocleidomastoid muscle form a triangle with the clavicle at the base	Palpate the clavicle bone.	1. Palpate the anterior-superior iliac pubic tubercle that delineate the course of inguinal ligament. inguinal ligament.
2. Internal jugular vein lies within the carotid sheath just beneath the apex of this triangle.		2. Palpate the femoral artery that runs beneath inguinal ligament.
3. The carotid artery also lies within the carotid sheath just medial and deep to the internal jugular vein.		3. Femoral vein lies ~1 cm medial and parallel to the femoral artery.

L. Wash hands.

M. Put on a sterile gown and gloves.

N. Create a sterile field; apply a sterile cover to the ultrasound probe if using during the procedure.

O. Open a sterile central venous catheter kit.

P. Prepare the area for cannulation using povidone-iodine for infants <2 months, chlorhexidine for infants and children ≥2 months of age as per institution policy; allow area to dry.

Q. Administer local anesthetic.

R. Flush the needle and catheter with sterile saline or heparinized saline solution, according to manufacturer's recommendations.

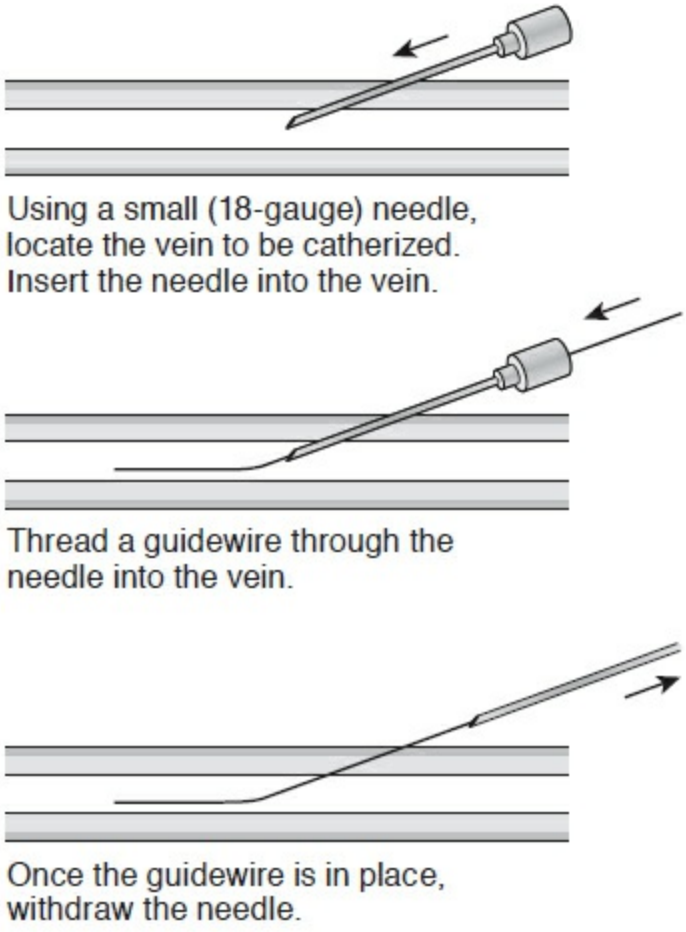
1. With or without ultrasound guidance, insert the needle with an attached syringe at the specified angle and depth, while gently aspirating with a syringe (**Table A12-2** and **Figure A12-1**).

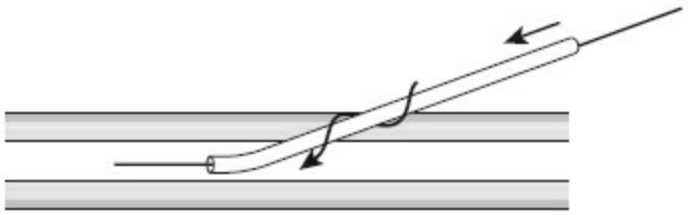
2. If using a neck approach, the internal jugular is preferred over the subclavian vein for successful cannulation and lower incidence of complications.

Table A12-2	ANGLE OF INSERTION BASED ON ANATOMICAL INSERTION SITE	
Internal Jugular Site	**Subclavian Site**	**Femoral Site**
Anterior Route: Aiming toward the ipsilateral nipple, insert needle at midpoint of the anterior margin of the sternocleidomastoid muscle at a 30° angle posteriorly.	Insert the needle under the clavicle bone at the junction of the medial and middle thirds of the clavicle, aiming toward the suprasternal notch at a 45° angle.	Insert the needle 1 finger's width below the inguinal ligament and medial to the femoral artery at a 30° angle, aiming toward the umbilicus.
Central Route: Aiming toward the ipsilateral nipple, insert needle at apex of the triangle formed by the sternoclavicular heads of the sternocleidomastoid muscle and the clavicle at a 30° angle.	The needle should be parallel to the frontal plane, directed medially and slightly cephalad, beneath the clavicle bone toward the posterior aspect of the sternal end of the clavicle.	

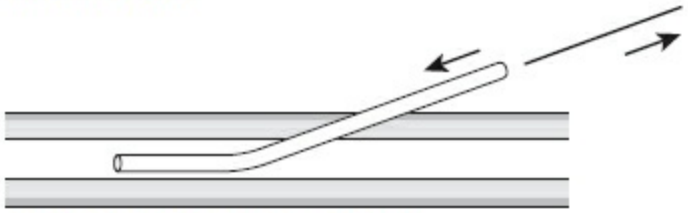
Posterior Route: Aiming toward the suprasternal notch, insert needle under posterior border of the sternocleidomastoid muscle, approximately halfway between the mastoid process and the clavicle.		
Depth of Insertion: Approximately 1-2 cm	Depth of Insertion: Approximately 2-3 cm	Depth of Insertion: Approximately 1-2 cm

Figure A12-1. Modified Seldinger Technique


Using a small (18-gauge) needle, locate the vein to be catherized. Insert the needle into the vein.
Thread a guidewire through the needle into the vein.
Once the guidewire is in place, withdraw the needle.



Using a rotary motion, advance the catheter over the guidewire, into the vein.



Finally, remove the guidewire, leaving the catheter in place.

S. When backflash of nonpulsatile dark blood is noted, stabilize the needle, disconnect the syringe, and cover the needle hub with a gloved finger.

T. Remove the needle and exert pressure for 5 minutes if pulsatile bright red blood return is obtained on needle insertion.

U. Insert a guidewire through the needle during a positive pressure breath or spontaneous exhalation; minimal to no resistance should be met.

V. Monitor cardiac rhythm for ectopy when inserting the guidewire, because it may be long enough to reach the heart.

W. Make a small incision with the scalpel adjacent to the needle to slightly enlarge the puncture site and accommodate the dilator and catheter.

X. Remove the needle, while maintaining control of the guidewire.

Y. Pass the dilator over the needle to gently dilate the skin and subcutaneous tissue.

Z. Remove the dilator while maintaining control of the guidewire.

AA. Pass the catheter over the guidewire and advance the catheter to the predetermined landmark, while maintaining control of the guidewire.

BB. Remove the guidewire.

CC. Aspirate blood from all ports of the catheter.

DD. Once blood return is confirmed, flush the ports.

EE. Connect the distal port to the monitor to confirm the central venous position waveform (nonpulsatile).

FF. Suture catheter or use other securement device as appropriate.

GG. Apply chlorhexidine-impregnated patch for infants and children ≥2 months of age.

HH. Apply sterile occlusive dressing.

I I. Obtain the chest or abdominal radiograph to confirm proper positioning of the catheter.

J J. Attach intravenous fluids.

IV. PRECAUTIONS/COMPLICATIONS

A. Hemothorax

B. Pneumothorax

C. Chylothorax

D. Bleeding/hemorrhage

E. Venous thrombosis

F. Arterial puncture

G. Heart perforation

H. Air embolism

I. Infection

J. Dysrhythmias

K. Catheter or guidewire embolism

L. Local subcutaneous tissue, nerve, artery, or vein damage

HANDOFF MNEMONICS FOR TRANSPORT AND TRAUMA

1. Transport

SMEAC

- **Situation**
  - – Location of patient
  - – Patient:

  Past medical history

  Medications

  Allergies

  Fasting/NPO (nothing by mouth) status

  - – Pertinent clinical events
  - – Treatment(s)
  - – Response to treatment/interventions
  - – Advice (including preparation of escort/transport)
- **Mission**
  - – Interfacility transfer/Search and Rescue
  - – Goals:

  Retrieve

  Resuscitate

  Stabilize

  Transport
- **Expected** course/worst case scenario
- **Equipment**
  - – Personnel necessary for transport, including consideration for airway/breathing/circulation
  - – Intervention supplies:

  Standard ABCD equipment

Case-specific items (e.g., antibiotics, antivenoms, blood products, orthopedic devices)

– Monitoring

- **Administration** (i.e., coordination, team roles, documentation, and timing)
- **Communication and Chain of Command** (i.e., contact name and number for coordinator, referral site [remember shift changes, ensure handover], transport team)

PRE-RETURN:

Secure and preempt problems with **ABCD**.

– **A**irway/**B**reathing: rapid-sequence intubation equipment readily accessible

– **C** (**AEIOU**): functioning **a**ccess with extension sets, **e**mergency drugs (vasoactive, anesthetics, etc.), (**i**nvasive) monitoring, **o**utput (i.e., indwelling catheters, Foley, naso- or orogastric tube), **u**nexpected problems anticipated (blood pressure derangements, defibrillator/pacing pads in place)

– **D**ocumentation: imaging, results

– **E**scort(s)

2. Trauma

MISTO

- **M**echanism of injury
- **I**njuries sustained
- **S**igns and symptoms
- **T**reatment(s) and response
- **O**bservation and vital signs
- **O**ther things:
  - Past medical history
  - Allergies
  - Medications
  - Fasting/NPO status

PEDIATRIC TRANSPORT FORM

MD, physician; PE, physical examination; T, temperature; HR, heart rate; RR, respiratory rate; BP, blood pressure; Hx, history; NICU, neonatal intensive care unit; ACLS, Advanced Cardiopulmonary Life Support; PICU, pediatric intensive care unit; ER, emergency room; ETA, estimated time of arrival

PEDIATRIC TRANSPORT FORM

Date:_____ Time:_____

NAME: _____ AGE: _____ WEIGHT: _____

DIAGNOSIS: _____

REFERRAL HOSPITAL: _____

REFERRAL MD: _____REFERRAL HOSPITAL PHONE: _____

ACCEPTING MD: _____

MEDICAL CONTROL MD: _____

HISTORY & PE:

VITAL SIGNS: T: _____HR: _____ RR: _____ BP: _____ Sp_{O_2}: _____ F_{IO_2} : _____

Hx:

PE:

Labs:

THERAPY/RECOMMENDATIONS:

Discuss possible interventions:

Discussion with transport team:

Medical Control MD:

BED CONTROL: Time contacted _____ Approved by_____ Time_____
(Name)

TIME TEAM DISPATCHED: _____ ☐Helicopter ☐ Fixed Wing ☐ Ground Ambulance

☐ Pediatric/NICU Specialty Team ☐ ACLS Ambulance

TIME ACCEPTED: _____ for admission to ☐ PICU ☐ ER ☐ PEDS FLOOR ☐ NICU

TIME ACCEPTING MD NOTIFIED OF ETA _____

ARTERIAL CATHETER INSERTION

I. INDICATIONS

A. Hypertension, hypotension, labile blood pressure

B. Continuous hemodynamic monitoring

C. Monitoring of vasoactive agents (vasopressors, vasodilators, or other cardioactive medications)

D. Frequent sampling of laboratory specimens (arterial blood gases or otherwise)

II. EQUIPMENT

A. Arterial catheter kit with appropriate-sized safety cannula over needle or procedure tray with catheter over guidewire (generally 22- to 24-gauge for radial and 20-gauge for femoral)

B. Ultrasound may be useful to locate artery, increase success rate, and reduce number of attempts

C. 2% chlorhexidine, 10% povidone-iodine, or 70% alcohol

D. Sterile towels, gloves, and gown

E. Mask

F. Protective eyewear

G. Surgical hat

H. Tape

I. Gauze sponges (2 x 2 and 4 x 4)

J. Arm board, if radial artery is being used

K. 3.0 or 4.0 suture and needle driver or other securement device (if not included in arterial catheter kit)

L. Transparent semipermeable dressing

M. Local anesthetic, such as lidocaine

N. Medication for sedation, if necessary

O. Pressure transducer, tubing, and pressure monitor

III. TECHNIQUE

A. Ensure that child and family understand procedure and all questions are answered and obtain consent

B. Gather supplies.

C. Assure intravenous access.

D. Wash hands.

E. Perform final patient identification process per institution-specific policy (i.e., time-out).

F. Ensure appropriate cardiorespiratory monitoring.

G. Perform modified Allen test (**Table A15-1**), if using radial artery.

Table A15-1	THE ALLEN TEST

The Allen test is used to test blood supply in the hand. It is performed prior to radial artery blood sampling or cannulation.

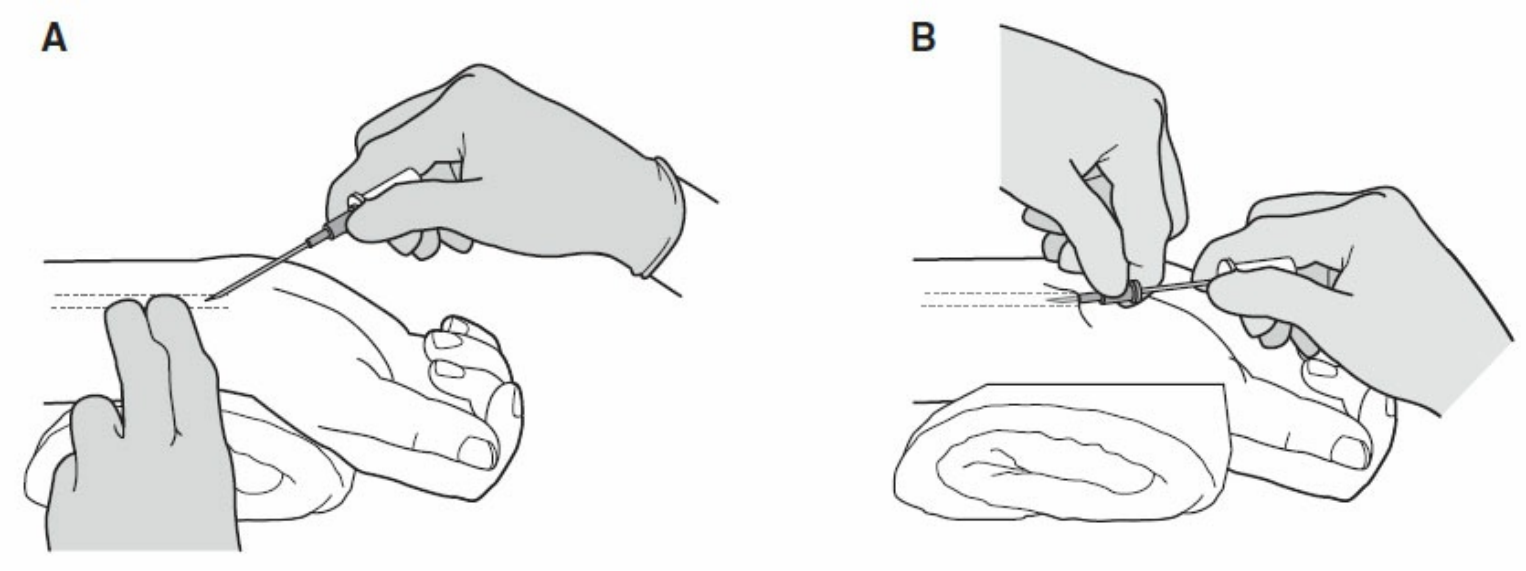
1. The hand is elevated and a fist is made for approximately 30 seconds.
2. Pressure is applied over the ulnar and radial arteries so as to occlude both simultaneously.
3. While elevated, the hand is opened. It should appear blanched.
4. Ulnar pressure is released, and the color to the hand should return in approximately 7 seconds.
5. If color does not return or returns after >7 seconds, the ulnar artery supply to the hand is insufficient and the radial artery cannot be safely punctured.

H. Use arm board for immobilization, if using radial artery.

I. Administer analgesia and/or sedation, as appropriate.

J. Position patient appropriately (**Figure A15-1** and **Table A15-2**).

Figure A15-1. Catheter-Over-Needle Technique



A) After extending and immobilizing the wrist, the radial artery is localized by palpation. The needle is inserted at a 20° to 45° angle. **B**) After the artery is entered, the catheter is advanced over the needle and the needle is withdrawn.

Table A15-2	ANATOMICAL POSITION FOR INSERTION[a]
Radial Artery	**Femoral Artery**
Position patient appropriately ○ Dorsiflex wrist 45-60° using small roll under the wrist	Position patient appropriately ○ Place towel or blanket under hips and place in frog-leg position

[a]Axillary, dorsalis pedis, posterior tibial, and umbilical artery catheterizations have been described but will not be discussed here.

K. If available and considered helpful, use ultrasound to identify vessels that may be appropriate for cannulation.

L. Obtain mask, cap, gown, and sterile gloves.

M. Prepare site using 2% chlorhexidine for infants >2 months of age, 10% povidone-iodine, or 70% alcohol.

N. Scrub site with back-and-forth motion using friction (or as per manufacturer's instructions); allow area to air dry for prescribed period.

O. Prepare sterile field, including use of sterile sheath on ultrasound probe if this equipment will be used during process of cannulation.

P. Identify landmarks; palpate artery.

Q. Infiltrate area with local anesthetic.

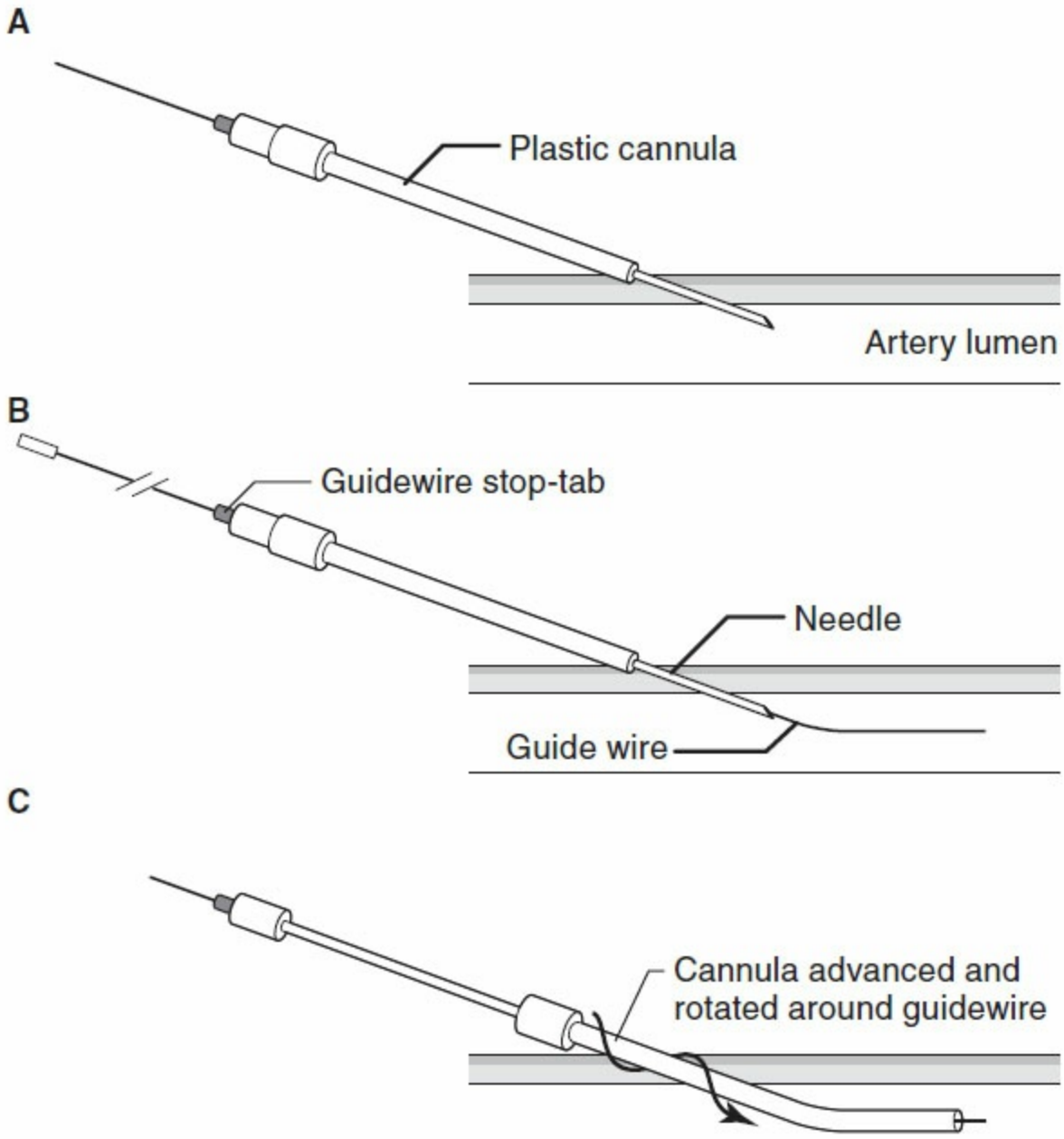
R. Puncture anatomical insertion site (**Tables A15-3**, **A15-4**, and **Figure A15-2**).

Table A15-3	ANATOMICAL INSERTION SITE
Radial Artery	**Femoral Artery**

◦ Puncture skin at a 30° angle, directing the safety catheter or needle toward the underlying artery. ◦ Advance the needle slowly, while watching for flashback. ◦ When blood is obtained, remove syringe and bring needle down against skin.	◦ Puncture skin at 45° angle, needle bevel up, below the inguinal crease and a few centimeters distal to palpable pulse. ◦ Enter through skin, advancing introducer needle into femoral artery. ◦ When blood is obtained, remove syringe and bring needle down against skin.

Table A15-4	INSERTION TECHNIQUE
Catheter Over Needle	**Modified Seldinger Technique**
Advance the catheter using a rotating motion to the hub and remove needle	◦ Immobilize needle with free hand. ◦ Advance guidewire through needle slowly; do not force wire in if resistance is. ◦ Remove needle and wire if unable to remove wire from needle during insertion attempts. ◦ Remove needle while holding guidewire in place. ◦ Pass catheter over guidewire while inserting catheter to the hub; do not force catheter if resistance is met. ◦ Remove guidewire. ◦ Secure catheter to skin after connecting transducer tubing. Ensure that the catheter is not inadvertently dislodged prior to securing with tape or suture material.

Figure A15-2. Modified Seldinger technique



A) The needle is inserted into the artery. **B**) The guidewire is advanced until the stop-tab reaches the needle hub. **C**) The catheter is advanced over the guidewire into the artery.

S. Attach catheter to transducer to observe for presence of arterial waveform and for ease of flushing and aspirating blood from catheter.

T. Secure with suture or sutureless securement device.

U. Surround site with chlorhexidine-impregnated sponge, per institution-specific protocol.

V. Cover site with semipermeable transparent dressing, per institution-specific protocol.

W. Dispose of supplies appropriately.

X. Remove personal protective equipment.

Y. Assess circulation to distal extremity.

Z. Wash hands.

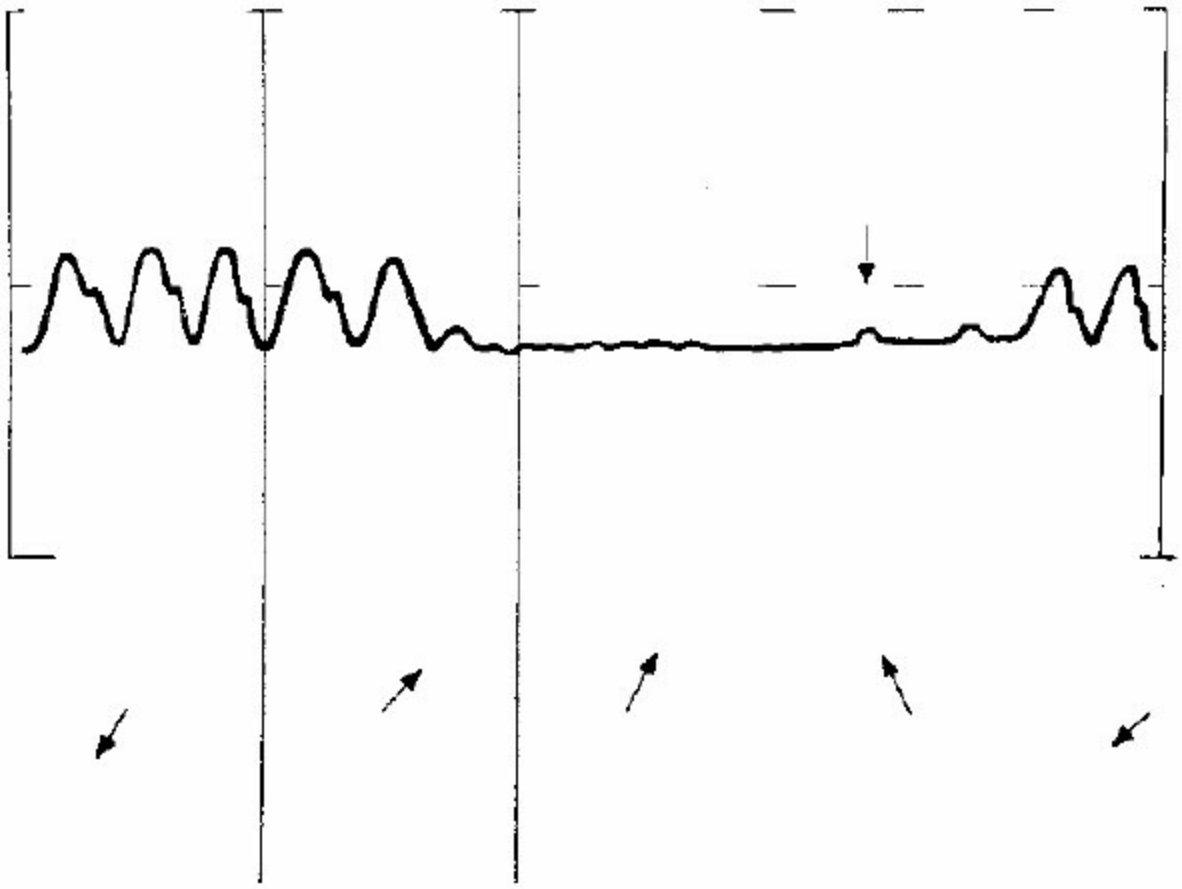
AA. Documentation of procedure may include the following:

1. Type of procedure
2. Informed consent
3. Risks/benefits
4. Skin preparation
5. Insertion site
6. Use of local anesthetic or systemic sedation/analgesia
7. Condition of child at end of procedure
8. Unexpected outcome

IV. VALIDATION OF MEASURES AND SYSTEM CHARACTERISTICS

A. Systolic return-to-flow blood pressure assessment (**Figure A15-3**)

Figure A15-3. Instructions for Obtaining Systolic Return-to-Flow Blood Pressure



A) Observe arterial waveform. **B**) Place a manual blood pressure cuff on the same extremity as the radial or dorsalis pedis arterial catheter and inflate it. Arterial waveform flattens as the cuff is inflated. **C**) Release pressure in cuff slowly until the first evidence of pulsatile waveform is again observed, at which point the pressure on the sphygmomanometer is noted. This

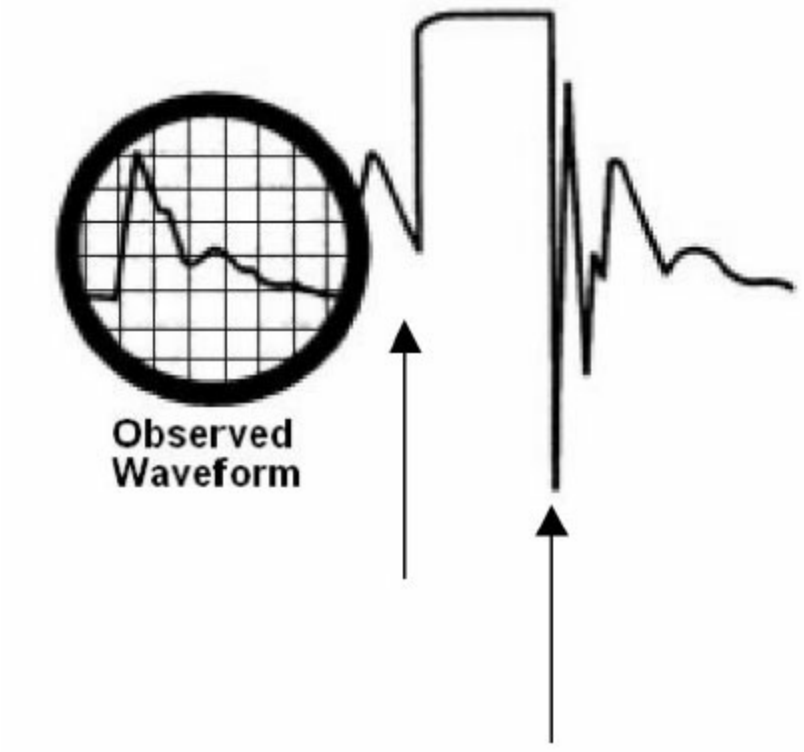
sphygmomanometer pressure is the return-to-flow or “true” systolic blood pressure.

1. Observe arterial waveform.
2. Place a manual blood pressure cuff on the same extremity as the arterial catheter and inflate until the arterial waveform flattens.
3. Release pressure in cuff slowly until the first evidence of pulsatile waveform is again observed, and note pressure on sphygmomanometer.
4. Sphygmomanometer pressure is the “true” systolic blood pressure and should correlate with pressure measured through the arterial catheter.

B. Square wave test

1. Use to test system characteristics of tubing (e.g., length, stiffness, presence of bubbles).
2. Rapidly flush the arterial line tubing (**Figure A15-4**).
3. Observe waveform for underdamping or overdamping (**Figures A15-5** and **A15-6**).

Figure A15-4. Square Wave Test



Application of a rapid in-line flush and the correlating evaluation of the monitoring system. First arrow indicates flush applied, and second arrow indicates the rapid frequency response when released. If a neonatal patient is using a pediatric transducer with an infusion pump, a square wave test is not possible using the flush device.
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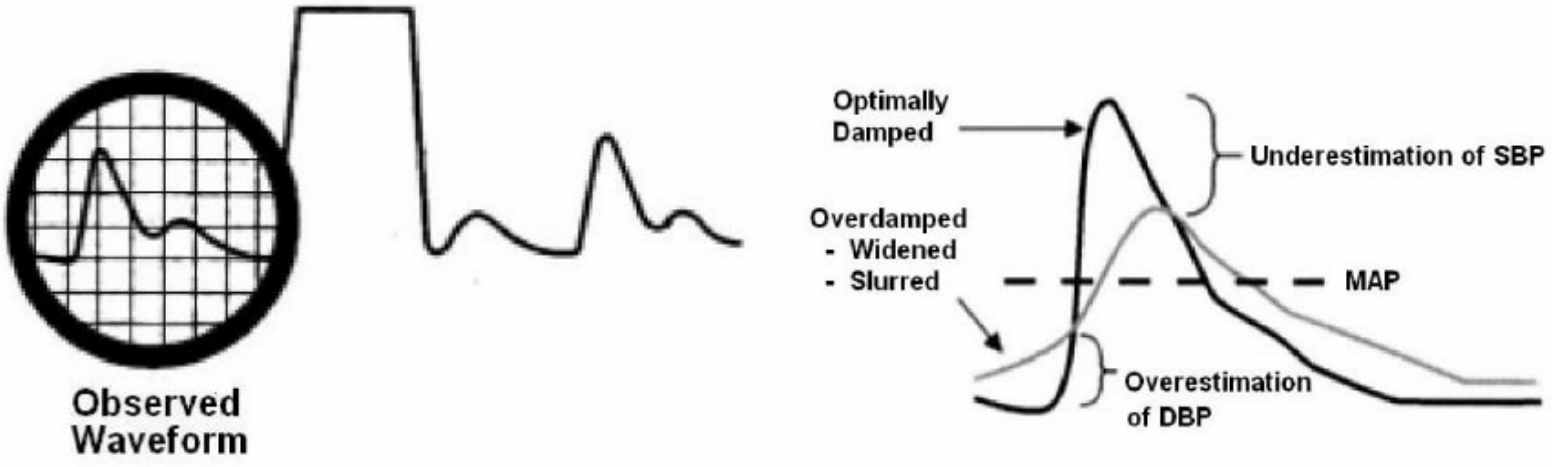
Figure A15-5. Overdamping



SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure.
An overdamped signal indicates loss of signal related to loose connections, air bubbles in the system, and/or inadequate fluid and pressure applied.
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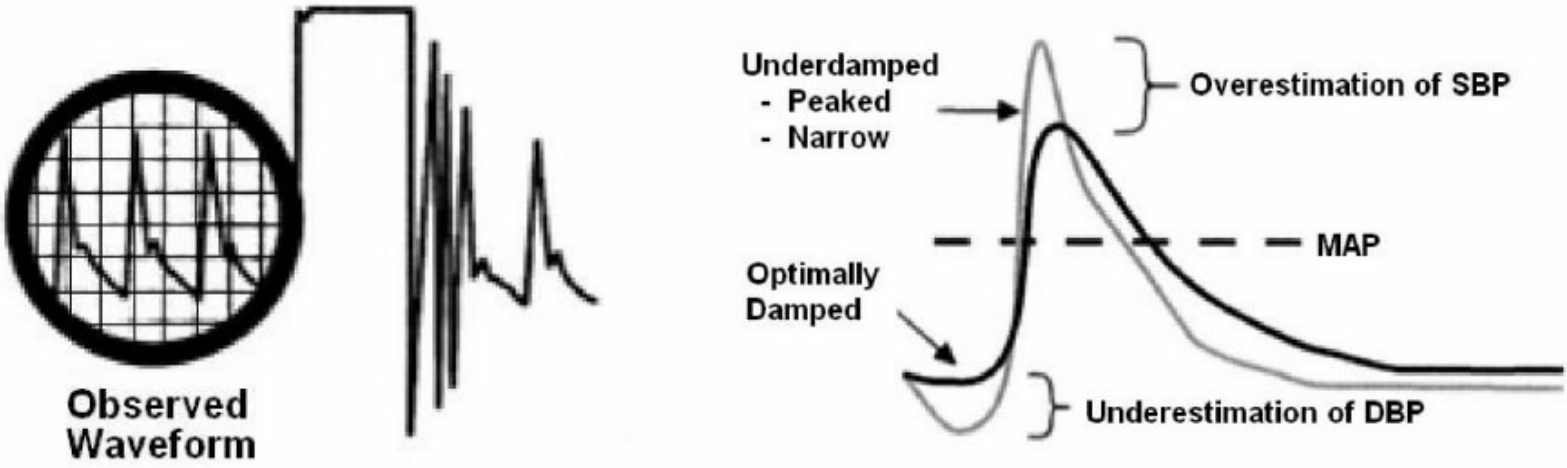
Figure A15-6. Underdamping



SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure.
An underdamped signal indicates significant increase in frequency response and is generally related to loss of calibration of the transducer, requiring a transducer change.
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C. Factors that may interfere with monitoring accuracy:

1. Connections of stopcocks and interface
2. Constant pressure application (300 mm Hg)
3. Elasticity alterations of the blood vessel wall
4. Reflectance of the pulse wave from the vessel walls or tubing
5. Air bubbles in the fluid column throughout the system

V. COMPLICATIONS

A. Compromised circulation/ischemia to extremity at insertion site and distal to insertion site

B. Infection

C. Hemorrhage

D. Arterial air embolism

E. Arteriovenous fistula

F. Arterial aneurysm

VI. PRECAUTIONS/CONTRAINDICATIONS

A. Ischemia of the extremity

B. Infection at the puncture site

C. Raynaud disease

D. Prior vascular surgery or cutdown procedure involving the artery to be punctured